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CONTENTS

	PAGES
Diseases Other than Tuberculosis (*concluded*)	89–91
Prognosis	92–92
Epidemiology (Incidence, Mortality)	92–93
Aetiology	93–94
Tuberculosis of Infancy and Childhood	94–97
Prevention (*BCG*)	97–103
Pathology	103–112
Anatomical	103–104
Bacteriological (*tubercle bacillus*)	104–107
Immunological (*tuberculin*)	107–110
Physiological (*chemical*)	110–112
Symptoms and Signs	112–114
Diagnosis	114–115
Tuberculosis of Organs Other than the Lungs	115–119
Treatment	119–128
General	119–121
Surgical (*collapse therapy*)	121–128

Primary Carcinoma of Lungs.—Those who have studied primary pulmonary cancer are familiar with the almost kaleidoscopic clinical manifestations of this malignant disease. The gross anatomy, as in cancer of other organs, will rarely furnish irrefutable criteria as to the particular histological structure which gave origin to the growth. Finally, the microscopic features of a fully developed pulmonary tumor will point to its histogenesis in exceptional cases only, the morphology of the neoplastic cell varying from one tumor to another and from area to area in the same tumor. The arrangement of the cells is, on the contrary, rather uniform, in most instances being that of adenocarcinoma. Pässler (1896) and Adler (1912) hold that carcinoma starting from the pulmonary alveolus is extremely rare or not proved, whereas Dömeny (1902) and Beitzke (1923) believe that certain forms of pulmonary cancer are of alveolar origin. The author of this article is convinced that the cells lining the air-sacs do not give rise to carcinoma because these cells are of mesenchymal origin and therefore could not originate epithelial malignant disease. Excluding the alveoli as a possible source of carcinoma, there remain to be considered the mucous glands and the bronchi. The former are structures that lie underneath the bronchial *membrana basalis*, and since they are made up of epithelial cells they are theoretically considered as being liable to transformation into an epithelial malignant new growth. But no worker has ever observed an early cancer of this kind, and the criteria, such as the glandular structure as well as the presence of mucus which are claimed to be characteristic of these neoplasms,

in reality distinguish a great many tumors originating from organs which normally form no mucous material. All the evidence points toward the conception that primary carcinoma of the lungs is only bronchogenic in origin. Apparently a malignant disease (primary) in the lungs results from the "cancerization" of the somatic cell. It is still uncertain whether a fully differentiated epithelial cell is liable to become cancerous. One of the factors leading to the development of a cancer is chronic inflammation, namely, irritation which causes degeneration of cells, eventually followed by excessive regeneration. In the lungs, however, the columnar epithelial cells lining the bronchi have never been observed to be in a state of regeneration as evidenced by mitoses and proliferation. The lining of the bronchi and their divisions is made up of three different types of cells: columnar ciliated cells, goblet cells and finally basal cells, a variety of small oval cells having a narrow cytoplasm and a nucleus rich in chromatin. The last mentioned cells lie close to the basal membrane; they do not form an uninterrupted syncytium as is observed in the skin, but are irregularly scattered here and there, forming cellular agglomerations. In the respiratory tract the process of regeneration takes place by virtue of the just mentioned "basal" cells, which are apparently endowed with latent developmental potentialities. In chronic bronchopulmonary diseases accompanied by damage of the bronchial epithelium, one often notices that instead of the ciliated epithelium there appear cuboidal cells superimposed by many layers of "transitional" epithelial cells, which have originated from the preëxisting "basal" cells. These cells also have a tendency to invade the pulmonary alveoli, thus giving the impression that the air-sacs are normally lined by cuboidal epithelial cells. They also not infrequently invade tuberculous cavities, where they ultimately become cancerous. The evidence is that cancer is always preceded by a process of regeneration. The development of epithelioma in areas subject to roentgen rays, smokers' epithelioma and tar and paraffin cancers is widely known. Observation has shown, too, that cancer of the lungs usually follows a long-standing chronic inflammatory process, such as tuberculosis, bronchiectasis, or pulmonary syphilis. These diseases may cause damage to the bronchial mucosa which is followed by excessive (pathological) regeneration that eventually leads to the development of an epithelial malignant disease. The apparent lack of any activity in the process of bronchial repair on the part of the ciliated columnar epithelium in the presence of an active "basal" cell proliferation seems to favor the conception that only the latter cells are concerned in the genesis of an epithelial malignant disease in the lungs. Postmortem material shows that a large percentage of all pulmonary tumors are of the basal or the squamous type. Since normally such cells are absent in the lungs, these tumors were said to have resulted from a conversion of the ciliated columnar into the squamous epithelial variety, a condition designated as metaplasia. This hypothesis has, however, not been borne out by close observation. It now seems probable that the "basal" cells already referred to, which lie close to the *membrana basalis* of the bronchus, are the progenitors of the stratified squamous epithelium. The peculiar differentiation of these cells into squamous epithelium is interpreted in the light of the ontogenesis of the tissue in question. From an embryological standpoint the tracheobronchial tree and the oesophagus represent two sister organs, and their development goes parallel. There is evidence that in the earliest stages of development the oesophagus is lined with one layer of cuboidal cells, which at the fifth week becomes doubled and at the tenth acquires goblet cells and ciliated columnar epithelial cells. Whereas in the bronchi the development ends at this phase, in the oesophagus these cells degenerate and desquamate, being subsequently replaced by a transitional epithelium and finally by a stratified squamous epithelium. It is assumed, then, that in pathological processes the bronchus, in the regenerative attempt, merely reaches in adult age the stage which the oesophagus has attained as an embryo. The process is, therefore, not a transformation of the adult columnar epithelium into a squamous type, but a development of undifferentiated cells, followed by proliferation and differentiation. The phenomenon is consequently not that of metaplasia, but is one of protoplasia (indirect metaplasia). The process of repair or regeneration in the bronchi is performed by the bronchial "basal" cell only. In physiological repair, these cells differentiate merely into the normal lining of the bronchus. But when the process is pathological, their fate depends in all probability on the nature of the stimulus. Thus, they may differentiate into metaplastic islands and so remain indefinitely, or they may develop into a malignant condition. In fact, most pathologists regard the phenomenon of metaplasia as being a precancerous stage. Two cases of primary carcinoma of the lung are reported, one a basal-cell cancer and the other a typical keratinizing epidermoid carcinoma. In the former, the tumor was sharply demarcated from the pulmonary parenchyma and originated from a division of the left bronchus. The tracheal, tracheo-

bronchial and mediastinal lymph nodes showed no tumor invasion. The other organs did not reveal the presence of any new growth. The microscopic examination showed the tumor to be composed of polyhedral cells having a large cytoplasm and a voluminous vesicular nucleus. These were arranged in columns and strands from 2 to 3 cells broad, which showed anastamosis and branching. Like most basal-cell cancers of the skin, this tumor did not metastasize. In the second case, the differentiation of the "basal" cells went one stage further, leading to a typical keratinizing of squamous cell carcinoma. Here the right lung parenchyma was found to be markedly invaded from the primary bronchus, with metastasis in the pleura, left lung, heart, regional lymph nodes and oesophagus. The microscopic sections showed a tumor without any definite arrangement, with nests or masses of cells scattered over the sections surrounded by thick bands of dense fibrous tissue. In the centre of the tumor mass, the cells had the typical appearance of the cornified epithelium encountered in the so called "pearls" of the epidermoid carcinoma. These keratinized masses were seen in practically all sections. *Conclusions: 1:* Carcinoma originating primarily in the lungs is bronchiogenic. *2:* The disease probably results from a pathological (excessive) regeneration following chronic inflammation of the bronchial tree. *3:* Of the cells lining the bronchial mucosa only the "basal" cells are concerned in the process of regeneration of the bronchial mucous membrane. It is assumed, therefore, that these cells likewise serve as a sole matrix for primary bronchiogenic tumors. *4:* Primary squamous cell epitheliomata and basal-cell epitheliomata of the lungs do not result from metaplasia of the preëxisting ciliated columnar epithelium, but originate through protoplasia (indirect metaplasia) of the undifferentiated basal cell of the bronchial mucous membrane.—*Primary Carcinoma of the Lungs, III. Histogenesis amd Metaplasia of Bronchial Epithelium, B. M. Fried, Arch. Path., July, 1929, viii, 46.*—(*J. S. W.*)

Fresh Tuberculous Foci and Carcinomatosis.—The author reports a case of terminal generalized tuberculosis occurring in association with a widespread metastasizing mammary carcinoma.—*Über gleichzeitiges Bestehen frischer tuberkulöser Herde und disseminierter Carcinommetastasen, F. Sturm, Deutsch. Ztschr. Chir., ccix, 406.*—(*H. J. C.*)

Antagonism between Tumors and Tuberculosis.—Carcinomata may develop in old tuberculous lesions, such as skin cancers on lupus, or pulmonary carcinomata in chronic pulmonary tuberculosis. Metastases of cancer and tuberculosis are, at times, found in the same lymph node. The antagonism claimed by Rokitansky to exist between the two diseases cannot be absolute. Experimental work gives evidence of some such antagonism. Rous's tumor material was inoculated into 80 animals. In 53 cases the tumor developed well and kept on growing until the animal died. Out of the other 27 cases the tumor did not grow at all in 8 cases; it grew only for a limited time in 16 cases, and developed poorly all the time in 3 cases. In the 53 successful transplants, no animal had tuberculous lesions, but in 23 out of the 27 failures tuberculosis was present. In 9 cases of tuberculosis the implanted tumor was completely scarred. In 5 cases of tuberculosis, typical, but small and retrogressive, Rous's tumors were found. Spontaneous immunity against this strain of tumor is exceedingly rare. On the other hand the presence of tuberculosis inhibited the growth of this tumor to varying degree in every case. This antagonistic relation is further proved by the observation that complete inhibition occurred in animals with the oldest and most extensive tuberculous lesions. The inhibitory effect of tuberculosis is not dependent on cachexia, and it is not a direct action of the bacilli on the tumor cells, since A. Fischer kept an *in vitro* culture of Rous's cells, infected with tubercle bacilli alive for more than a year. The cells were not damaged by the bacilli; the latter were phagocytosed but not killed by the tumor cells.—*"Antagonismus" zwischen Roustumor und Tuberkulose?, O. Teutschlaender, Klin. Wchnschr., August 27, 1929, viii, 1606.*—(*M. P.*)

Fibrosarcoma of Mediastinum.—A white male, aged forty-two, complained of pain in the right shoulder of four month's duration, associated with loss of eight pounds in weight and, for one month, unproductive cough. Stereoroentgenographs of the chest showed an ovoid dense mass with sharply defined borders in the lower vertebral position of the right lung field about 9.5 x 13.5 cm. in diameter lying close to the posterior chest-wall between the eighth and eleventh ribs. At operation, a large, hard, encapsulated mediastinal tumor about the size of an orange was shelled out from its capsule. The patient died of shock shortly after the operation. *Pathological diagnosis:* Fibrosarcoma.—*Fibrosarcoma of the Mediastinum, S. A. Robins, Am. J. Roentgenol. & Rad. Therapy, October, 1929, xxii, 351.*—(*W. I. W.*)

Prognostic Tests.—Only serial sputum examinations are of value in drawing prognostic conclusions that an increase of tubercle bacilli is unfavorable while a decrease indicates a favorable course of the tuberculosis. The finding of Much's granules is viewed as prognostically favorable. The sedimentation rate of the erythrocytes is only definitely of value prognostically in so far as a positive finding does not with certainty indicate pulmonary tuberculosis and a negative does not exclude it. More valuable from a prognostic standpoint is the reaction of Gerlocy. A negative plasma colloid-lability reaction, according to Gerlocy, definitely excludes an active pulmonary tuberculosis, while a positive reaction in the absence of other factors causing such reaction verifies the existence of pulmonary tuberculosis. In addition, the grade of the reaction can be correlated with the activity and inactivity of the process as well as indicating its exudative or productive character.—*Über den prognostischen Wert der Sputumuntersuchung, der Senkungsgeschwindigkeit der Erythrocyten und der Plasmakolloid-Labilitätsreaktion von Gerlocy bei Lungentuberkulose, J. v. Szaboky, Zeitschr. f. Tuberk., 1928, l, 413.*—(*H. J. C.*)

Prognostic Tests.—The urochromogen test was found more sensitive than the diazo reaction. A positive finding must be viewed as unfavorable prognostically and should caution against the use of irritation therapy. The combined testing of allergy, red-cell sedimentation and the differential blood picture were found valuable. The favorable outcome of these tests indicates a favorable prognosis and the possibility of proceeding more accurately therapeutically.—*Über den Wert der Urochromogen- und Diazo-reaktion, sowie den des Blutbildes, der Senkungsreaktion und der Allergieprüfung für die Beurteiling der Kehlkopftuberkulose, L. Rickmann and F. Ernst, Zeitschr. f. Tuberk., 1928, l, 465.*—(*H. J. C.*)

Prognosis of Cavities.—The author reports the outcome of cavity cases during the course of 2 to 17 years in a material of 626 cases, among which 489 were dead while 21.8 per cent still were alive. It is pointed out that during the first 5 years after taking the cure 75.7 per cent of cavity cases were dead. Cases that lived beyond these 5 years have a favorable prognosis and healing may occur. In none of the cases was operative therapy utilized.—*Zur Kavernenprognose des Industriearbeiters, E. Sprungmann, Zeitschr. f. Tuberk., 1928, li, 432.*—(*H. J. C.*)

Tuberculosis Mortality in Belgium.—The statistics available are, unfortunately, incomplete, and prior to 1870 are generally useless. Unfortunately also, their basis has been modified since they were commenced and hence they are not always comparable. From 1870 to 1887 deaths from pulmonary tuberculosis were merged with those due to other respiratory diseases. Nevertheless a series of graphic charts is available which is of considerable interest. From 1867 to 1890 the rural mortality from pulmonary tuberculosis rose while the urban rate fell abruptly. By 1900 the rural rate was slightly higher than the urban, in 1913 it fell below it, and thereafter the two continued almost parallel up to the War. At this time there was a recrudescence of the disease in all countries, but especially in the more populous communities. After the War there was again a decrease except in communities under 5,000 population. Communities of over 100,000, as Antwerp, Brussels, and Liége, also Ghent and Schaerbeek, show a high mortality for the period of the War, with a subsequent rapid descent after it, which has slowed up since 1921. Communities of 20,000 to 100,000 show a similar but less marked change, while those of 5,000 to 20,000 (about one-third of the total population) show the lowest mortality of all. It is well known that the less important centres have the largest proportion of children and old people, among whom the tuberculosis mortality is lowest. As to sex, in the larger cities the masculine rate has always been above the feminine. In the smaller communities, however, the reverse was formerly true, but now the two are nearly the same. *Conclusions:* In a general way the mortality from pulmonary tuberculosis is highest in the large centres of population. It is, however, lowest in communities of 10,000 to 20,000. In the smallest communities it is no longer decreasing, likewise in those between 20,000 and 100,000. For those over 100,000 it is higher in males, but in the smaller centres has been about the same for both sexes since 1890. Prior to this there was a higher female mortality. Numerous graphs are appended.—*Contribution à l'étude de l'évolution de la mortalité tuberculeuse en Belgique, E. Olbrechts, Rev. Belge d. l. Tuberc., January-February, 1929, xx, 24.*—(*A. P.*)

Tuberculosis in Interior East Africa.—In the interior of east Africa tuberculosis occurs mostly in adult life at the time the natives leave their homes for employment in the cities, on plantations and on the coast. The disease is not necessarily of serious prognosis, for good attention and food, etc., usually lead to cure. It need not be feared that the disease will spread or reach epidemic proportions in central east Africa, for it

appears that a slow, gradual infection of the population is taking place. The natives in central regions have good food and are a healthy people. One hundred and three adults and 51 children (up to 15 years old) were tested with tuberculin. Of the adults ten reacted and of the children only one reacted. (He was four years old and his mother also had a positive reaction. The father had died of haemoptysis.) None was clinically sick.—*Beobachtungen über Tuberkulose unter den Eingeborenen im Innern Ostafrikas, F. K. Kleine, Deutsche med. Wchnschr., January 24, 1930, lvi, 130.*—(*H. S. W.*)

Nursemaids and Teachers as Sources of Tuberculous Infection.—The author deplores our lack of control over such possible sources of infection as nursemaids, governesses, and kindergarten-teachers. He cites the case of a kindergarten teacher who came to California 15 years ago. Tubercle bacilli were found in her sputum at that time. After a few years she was employed by a private kindergarten. She taught at this school for ten years, then died of tuberculosis in a sanatorium. Another instance was that of a family of two children, ten months and three years of age, respectively. A nursemaid was employed in June, 1929, and dismissed in October, 1929. In November the three-year-old child died of tuberculous meningitis. The other child also has a pronounced infection, and the outlook is doubtful. The nursemaid was examined and an active tuberculosis discovered. In another family, a child seven months of age died of miliary tuberculosis after an illness of three weeks. When the child was one month old, a nursemaid was employed who remained with the family for one month. This nursemaid died of tuberculosis within a few days of the child's death. It is inconsistent to demand tuberculin-testing of our dairy cattle and yet permit occurrences of this sort. We must demand a health examination of all persons making it their business to care for or help care for infants and children. Only five states are known to have laws requiring teachers to be free from tuberculosis. Registration of all nursemaids, governesses, and kindergarten-teachers should be required, as should also periodic examinations of these persons.—*Tuberculosis in Infancy and Childhood: Nursemaids, etc., as Sources of Infection, H. Dietrich, Arch. Pediat., March, 1930, xlvii, 197.*—(*P. J. W.*)

Milk and Tuberculosis in Infants.—Milk is an excellent culture medium for many pathogenic organisms, of which the tubercle bacillus is one of the most important. The Massachusetts tuberculosis clinics being held in the schools throughout the state under the auspices of the Department of Public Health have shown that there are 24 per cent noncontact reactors in urban communities as against 36 per cent in rural ones. Why should the country-bred child, with advantages of fresh air and sunlight, be at this disadvantage? An important factor is that most cities safeguard their milk supplies, whereas few rural communities do. Theoretically a "tuberculosis-free herd" is safe as regards tuberculosis, but not always in fact, because dishonest cattle dealers "plug" the cattle they sell in the open market, that is, give them a dose of tuberculin before delivery which prevents their reacting to subsequent tests for about sixty days. If a cow must be bought from any but an accredited area, she should be quarantined and retested after ninety days before being admitted to the herd. Meanwhile her milk should not be consumed raw. A herd really kept free of tuberculosis helps to safeguard not only the consumer but also the herd itself, a point often overlooked by the farmer who complains. The second method of protection is by pasteurization, not always perfect even in honest hands. The recent Montreal typhoid epidemic is said to have been caused by milk from a pasteurizing plant where actually only about half the output had been so treated because of the limited size of the plant. Possible sources of error in honest hands are: *1:* Dead ends of piping in which milk remains unpasteurized and afterward recontaminates the whole product. *2:* Leaky valves in piping. Pipes should be broken beyond all such valves so that leaks can be detected. *3:* Collection of froth and foam at the top and its insulation by contained air from the pasteurizing temperature. This can be corrected by avoiding splashing in the vats and introducing live steam to the surface. *4:* Failure to heat the vats before introducing the milk, or too fast a flow in constant flow pasteurizers. *5:* Faulty thermometers. It is clear that the "best" milk is that from healthy cattle, handled by healthy individuals with scrupulous care, and conscientiously and intelligently pasteurized. On October 30, 1923, the Board of Health of Milton, Massachusetts, passed a regulation requiring that on and after April 1, 1924, only pasteurized milk or that from tuberculin-tested cattle retested every six months, should be sold in the town. Owing to the complaint of the farmers and the possibility of errors nullifying the results, a study of conditions was begun in July, 1927. One hundred babies and preschool children were tested in the Milton clinic and a similar

number in the Canton clinic (Canton, the next town, relies only on dairy inspection). The tests were performed intracutaneously with 0.1 cc. of 1–1000 tuberculin supplied by the Children's Hospital of Boston. Reactions were read after approximately forty-eight hours and considered positive if 5 mm. or more in diameter, negative if 2 mm. or less, doubtful in between. Milton showed 1 positive, 4 doubtful and 95 negative; Canton 7 positive, 12 doubtful and 81 negative. All seven of Canton's positive cases were known to have consumed raw milk at some time, and 9 of the doubtful ones, while none were known to have been exposed to human tuberculosis in either Milton or Canton, though 4 of the Canton cases had a family history of such. Recently the Massachusetts legislature had two bills before it. The last, House Bill No. 56, required pasteurization of milk not from tuberculin-tested herds, also either pasteurization or certification of any milk supply of 200 quarts per day or more. Both bills failed by an overwhelming majority although supported by the Department of Public Health. *Summary: 1:* One hundred children of approximately similar social status in adjacent towns were tested with 1–1000 tuberculin intracutaneously. *2:* Conditions in the two towns were similar except that 47 of the Canton children drank unboiled or unpasteurized milk, against 6 from Milton, and that all the Milton raw milk was from tuberculin-tested herds. *3:* The incidence of reactors in Canton was seven times that in Milton. *4:* In Canton all the 7 positive and all but two or three of the 12 doubtful fell in the raw milk group, while in Milton 2 of the 4 doubtful had been definitely exposed to tuberculosis. *5:* Despite the small size of the figures, the necessity for legislation along the lines of Massachusetts House Bill No. 56 of 1929 is clearly demonstrated.—*Tuberculosis Rate in Infants in Relation to the Milk Supply, P. R. Withington, New England J. M., November 21, 1929, cci, 1034.*—(*A. P.*)

Diagnosis of Tuberculosis of Tracheobronchial Lymph Nodes.—The diagnosis of tuberculosis of the tracheobronchial lymph nodes is of the greatest importance, because the entire future of the child depends on it. It may make necessary open-air or even preventorium life and careful attention to diet, because, if one is not careful, the child may develop generalized tuberculosis or tuberculous meningitis. One should not make the diagnosis of tuberculosis of the tracheobronchial nodes, as a rule, if only a positive Pirquet reaction and more or less confirmed clinical and roentgenological signs are present, and all the members of the family and those surrounding the family are healthy. On the contrary, one must strongly suspect it if the child has been in close contact with tuberculous parents or other people. Pediatricians have been too inclined to make the diagnosis of this disease on the sole basis of disputable roentgenological interpretations. The authors agree with Leon Bernard that pretuberculosis does not exist: a child is or is not tuberculous. The ease with which other conditions may be confused with tracheobronchial gland tuberculosis is stressed, as is also the risk of too much reliance on any of the clinical signs and symptoms. With regard to X-rays, only instantaneous roentgenography should be used, and pictures should be taken in the frontal and lateral positions. Those interpreting the films should have made pathologic controls in similar cases. Roentgenography in the right oblique anterior positions is not satisfactory because shadows cast by the pulmonary vessels may lead one to believe that a nonexistent adenopathy is present. The purpose of the lateral roentgenogram is to examine the intertracheobronchial group, which is not visible on anteroposterior roentgenograms. It is useless to examine a lateral roentgenogram without the anteroposterior roentgenogram.—*The Diagnosis of Tuberculosis of the Tracheobronchial Glands, P. Armand-Delille and C. Lestocquoy, Am. J. Dis. Child., December, 1929, xxxviii, 1125.*—(*P. J. W.*)

Tuberculin Skin Reactions.—The Mantoux, or intracutaneous, tuberculin skin test is recommended as being twice as sensitive as the Pirquet scarification test. To avoid a general reaction, the initial dilution should be 1:1,000. Stronger solutions may be used for later tests. The diluted solution should be kept on ice and should not be used after it is one or two days old. A control to the Mantoux test is not necessary. Tuberculin is not thermolabile, but remains active after boiling for several hours. Any acute febrile disease may suppress the tuberculin reaction. The results of tests on 7,668 children are given. These children were patients in the Children's Medical Division of Bellevue Hospital, New York, their ages being from birth to 13 years. The observations were made between January 1, 1921 and January 1, 1929. The first six months show about 3.38 per cent positive reactions; from 6 months to 2 years, 8 to 12.5 per cent; 2 to 6 years, 15.6 to 18 per cent; 6 to 10 years, when the contacts of the child increase, 26.3 to 29.7 per cent; and 10 to 13 years, 38.8 and 43 per cent. It is evident that there are only about half as many children infected at each age in this country

as in the first series published from Europe, and that not over 40 to 50 per cent of American children in the great cities harbor the bacillus even at puberty. Thus American children are not all infected by the time of puberty; and thus the tuberculin reaction is an important one. Infected children, who carry tubercle bacilli in their bronchial nodes or elsewhere, are the ones who need early correct diagnosis. The Mantoux test provides the means of making it. It should be used on every child who is underweight, anaemic or languid, or who has an unexplained irregular fever. These children with "tuberculous infection," so called, have true incipient tuberculosis, latent or active. Their disease is generally in lymphatic tissue, where it is easily encapsulated, and where scar tissue does no great harm. Under intelligent, watchful care, they usually do well.—*Tuberculin Skin Reactions, C. H. Smith, Am. J. Dis. Child., December, 1929, xxxviii, 1137.*—(*P. J. W.*)

Size of Intracutaneous Tuberculin Reactions.—It is generally agreed that the positive reactions obtained with artificial tuberculin are evidences of sensitiveness to protein, or true allergy, established some time after the initial infection with the tubercle bacillus. This study is made to determine whether any clinical significance could be attached to the size of the visibly reacting area, when single intracutaneous tests were performed under conditions similar to office practice, with tuberculin purchased on the open market. New patients entering the Stanford Children's Clinic (San Francisco) were tested with 0.1 mgm. tuberculin without reference to their complaints. Tests were read after 48 hours, and only those were considered positive in which each diameter of the hyperaemic area was 5 mm. or more. Seven hundred positive reactions were obtained. Elliptical and circular areas were calculated according to shape. The average size was 459 sq. mm. (about the area of a silver quarter dollar). The smallest reactions occurred in the first year group, and there was an increase in the size of the reactions with each added year of age up to the sixth. The oriental races gave considerably larger reactions than other races. Children who had had contact with tuberculous persons reacted more strongly than children who gave no history of contact. A relation between sensitivity and the extent of roentgenographic lung-markings is suggested. Children suffering from phlyctenular conjunctivitis, lymph-node, and bone and joint tuberculosis reacted more strongly than those with hilar disease, while the latter gave slightly stronger reactions than children suffering from an adult type of tuberculosis. The reactions of patients classified as "latent tuberculosis" were slightly less in size than the general average. The difference is not sufficient to relate the degree of sensitization to the degree of tuberculous activity. Much more can be learned by observing the patient over a considerable period, testing him from time to time.—*The Size of the Reacting Area in Intracutaneous Tuberculin Tests in Relation to Classification of Disease and to Other Clinical Factors, L. B. Dickey, Am. J. Dis. Child., December, 1929, xxxviii, 1155.*—(*P. J. W.*)

Tuberculin Reactions among Hospitalized Children in Iowa.—Data have been collected from the records of patients treated in the Children's Hospital, Iowa City. The children are preponderantly of the indigent class. Most of them come from small towns, or else directly from farms. Clinically recognized tuberculosis has been excluded. During the earlier period of the survey, 412 cases were tested by the cutaneous (Pirquet) method, with a percentage positive of 3.88. The maximum incidence was reached at 12 years, with 15.8 per cent positive. In 1,356 cases tested by the intracutaneous method, 16.59 per cent were found to be positive, the yearly incidence increasing irregularly to a maximum of 31.4 per cent at 16 years. Thus, at the age of 16, approximately one-third of the hospitalized children, excluding those with recognized tuberculosis, have become infected. The intracutaneous test is clearly more reliable as a diagnostic measure. A study was made of a group of children tested by both methods, with no significant amount of time elapsing between the two tests. In a group of 42 patients reacting positively to the intracutaneous test, only 5 had positive cutaneous reactions, a percentage of 11.9. Of 37 patients with clinical tuberculosis, 28, or 75.6 per cent, reacted positively to the cutaneous (Pirquet) test. Of 30 patients with clinical tuberculosis, 23, or 76.6 per cent, reacted positively to the intracutaneous (Mantoux) test. Thus there seems to be but slight difference in the adequacy of the two methods in cases of known active tuberculosis. The total incidence of tuberculous infection in children of rural districts is in general lower than that in children of congested districts.—*A Tuberculin Reaction Survey among Hospitalized Children in Iowa, M. H. Taylor, Am. J. Dis. Child., February, 1930, xxxix, 316.*—(*P. J. W.*)

Interpretation of Roentgenograms of Chest in Children.—This study was under-

taken to ascertain just what pathological changes were present in the lungs of children to account for shadows in the roentgenograms of the chest. A careful history of every patient was taken, with repeated physical examinations and the necessary laboratory work. Roentgenograms of the chest were made at intervals during life. If the patient died, films were taken of the chest postmortem, and finally films were made of the excised lungs in many cases. The study was made at the Children's Memorial Hospital in Chicago. The primary focus is found far more often in postmortem specimens than on the films. This is true, not only because of the location of the focus behind denser structures in some cases, or because shadows are not cast, but also because at times the primary focus may heal without leaving any noticeable evidence of its existence. Tuberculous cavities were found pathologically on several occasions, but in only a few instances was it possible to demonstrate them in the roentgenogram. The hilum is infected secondarily to the lung tissue, and this involvement is usually more extensive, remains larger, and leaves more recognizable scarring than the pulmonary infection. It is because of hilar shadows that many diagnoses of tuberculosis are made in error. Pathological change in the hilum may be due to many different processes, some of which for a time resemble those due to tuberculosis. In only about 50 per cent of the cases of miliary tuberculosis could the diagnosis be made from the roentgenograms. The Pirquet reaction was positive in 50 per cent of the cases of miliary tuberculosis. Calcification is a term frequently used by all who interpret roentgenograms, and many diagnoses of tuberculosis have been made on its supposed presence. The frequency of calcified nodes in the chests of children is overestimated. In the series reported, calcification, when present, was always considered tuberculous because of its presence in a definitely tuberculous process. It has been stated that it takes about three years for calcium to be deposited, but it has been found in children under two years of age. Calcified lesions in children are more important than those in adults because they frequently represent only partial healing, from which a disseminated tuberculosis may originate. More care should be taken in the diagnosis of calcification with its related diagnosis of tuberculosis. In 171 necropsies, the majority of which were performed on patients under five years of age, calcification was found in only five instances.—*Interpretation of Roentgenograms of the Chest in Children Based on Observations at Necropsy: Tuberculosis, J. A. Bigler, Am. J. Dis. Child., December, 1929, xxxviii, 1166.*—(*P. J. W.*)

Monocytes in Tuberculosis of Infants and Children.—In the diagnosis of active tuberculosis, too much emphasis cannot be given to the importance of a careful history and painstaking physical examination. Laboratory tests are of inestimable value to substantiate and supplement clinical findings but should not be relied on alone. Despite the development of numerous technical procedures within the past fifty years, mistakes in diagnosis continue, often from incorrect interpretation and too complacent reliance on the results of one or more tests. Roentgenological evidence alone is not sufficient, as many different pathologic processes cast shadows closely simulating tuberculosis. Tuberculin skin reactions determine the presence of infection, not its activity or extent. Persistent negative reactions with increasing doses are of great significance in excluding tuberculosis, but even here caution must be exercised to rule out anergic states (in extensive and overwhelming disease). It is well known that a careful study of the white blood cells is helpful in diagnosing many diseased conditions but until recently haematological studies have not been regarded as a dependable diagnostic or prognostic aid in human tuberculosis. Recent work of Sabin, Cunningham and Tompkins, Rogers and others, however, has shown that the monocyte-lymphocyte ratio and the presence of modified monocytes and epithelioid cells are of practical as well as academic interest in the diagnosis and prognosis of tuberculosis. The monocyte is more commonly known as the transitional or large mononuclear cell. Normally their number compared to that of the lymphocyte is about 1 to 4 or lower, but during periods of tuberculous activity is much higher. Blackfan and Diamond have, during the past year, studied the blood of actively tuberculous patients by means of the "supravital technique." In group 1 the clinical diagnosis was confirmed by necropsy, biopsy, guinea-pig inoculation, or the finding of tubercle bacilli, but in group 2 it was not so confirmed. In group 1, the peripheral blood of infants and children during the active phase showed both a relative and absolute increase in monocytes and a corresponding decrease in lymphocytes, with the appearance in the blood of modified monocytes and epithelioid cells. During healing and quiescence the reverse occurred. Similar findings were generally obtained in group 2, strongly suggesting the correctness of the diagnosis. These modified monocytes and epithelioid cells are readily recognizable and differentiated from other

pathological mononuclears by Sabin's "supravital" staining method, but cannot be positively identified by Wright's method. However, when in a fixed smear an unusual number of abnormal mononuclear cells appear, tuberculosis should be considered and confirmation sought. Two illustrative case histories are cited. *Conclusions:* In infants and children the degree of activity of the tuberculous process is indicated in the peripheral blood by (1) the ratio of monocytes to lymphocytes, and (2) the presence of modified monocytes and epithelioid cells ("supravital" stain). Such changes are of diagnostic and prognostic aid and may be used as a guide to treatment.—*The Diagnosis of Active Tuberculosis in Infants and Children, (The Significance of the White Blood Cells), K. D. Blackfan and L. K. Diamond, Meeting Vermont State Med. Soc., October 12, 1928, published in New England J. M., July 25, 1929, cci, 170.*—(*A. P.*)

Tracheotomy and Tuberculosis.—The author quotes Landouzy's assertion, made in 1899, that children who had undergone tracheotomy for laryngeal diphtheria were especially susceptible to tuberculosis, and that very few reached adult life. A review of the literature shows that the experience of most observers does not confirm Landouzy's observation. The case of a mother, aged 32, is reported, who was tracheotomized for laryngeal diphtheria at the age of two; also the case of her child, who was tracheotomized at the age of four. There was never any tuberculosis in either patient. The author takes sides with Landouzy's opponents.—*Tracheotomy and Tuberculosis, J. D. Rolleston, Brit. J. Child. Dis., July–September, 1929, xxvi, 200.*—(*P. J. W.*)

History of BCG.—One experiment of twenty years ago, since become classical, cast much light on artificial immunization against tuberculosis in cattle. If one selected two cows, one reacting to tuberculin, the other not, that is, one tuberculous, the other not, and on the same day inoculated into the veins of both the same quantity of virulent tubercle bacilli which ought to kill in four to six weeks, it was noted with astonishment that the healthy cow died within fifty days of massive miliary tuberculosis of the lungs, while the other continued in the appearance of health and the preexisting lesions did not seem to be aggravated. This experiment, together with some old observations of Marfan, for human beings, explained certain disturbing results observed on attempting Behring's bovovaccination in 1905. One notion regarding active immunity, until then unknown, was the necessity of the presence in the organism of tubercle bacilli, in order to confer resistance against new infection. The same notion explains the slowness of evolution of tuberculous disease in cattle, which may exist for years notwithstanding the appearance of health. Further experimental work clearly showed that living tubercle bacilli confer resistance to reinfection, at least in warm-blooded animals. Practical utilization of this knowledge was full of difficulty, as the tubercle bacilli of warm-blooded animals were all potentially pathogenic, while those of the cold-blooded ones were of too degraded a type to be effective in protecting mammals. The quality of the tubercle bacilli is of prime importance. The bacillus must preserve its essential character, with the exception of pathogenicity; it must be inoffensive and yet effective. *Development of BCG Strain:* A very virulent bacillus of bovine origin was selected, originally isolated by Nocard from tuberculous milk. On February 7, 1908, this was seeded for the first time on glycerinated potato bile. A medium containing bile was selected because of the observation that cultures of bacilli readily became reduced to a fine emulsion in a mortar if moistened with one or two drops of sterile ox-bile which seemed to have a saponifying action on their waxy coating. This led to the belief that cultures frequently renewed over a long period on such a medium might eventually become modified as to this fatty-waxy envelope. Growth on biliated potato medium at once yields a rich culture, the surface colonies being of a gray-green color. The first transplants were made every two weeks, and later every three weeks. On January 1, 1928, after twenty years, 347 successive transplants had been made without interruption. Modifications of virulence took place very slowly, but gradually. At the end of 20 transplants the inoculation of 0.001 mgm. intraperitoneally in the guinea pig, provoked in a week's time enormous mesenteric lesions, riddled with masses of tubercle bacilli, and the spleen, enlarged to three times normal size, showed many. The same dose injected intravenously in the rabbit killed the animal in two to three weeks by septicaemia. In the young calf, on the other hand, the original lethal dose of 3 mgm., injected intravenously, produced only chronic lesions. By the thirty-fourth transplant the biliated bacillus no longer caused lesions in cattle and immunization experiments with these animals were begun. The horse and the ass, however, behaved like the smaller laboratory animals in their reactions, 3 mgm. of the biliated bacilli intravenously causing death from acute miliary disease in twenty-five to fifty days. In

1919, after the 198th transplant, the bacillus grown on glycerinated potato medium looked like a normal culture and tuberculin was detectable in new cultures from the first. Its lack of virulence, however, was complete in doses of 1 mgm. for the guinea pig and rabbit, 5 mgm. for the horse, and 100 mgm. for the ox. Doses of 20 mgm., given intravenously to five or six months' old calves, caused no immediate reaction, but, beginning with the second week, a violent hyperthermia, with intense constitutional phenomena,—anorexia, wasting, etc., —suggesting a crisis of "typho-bacillosis." This lasted four to eight days, followed by resolution and complete restoration to health, with never the least development of lesions. Doses of 50 to 100 mgm., inoculated under the skin of the dewlap of young cattle, caused in forty-eight hours an intense oedema, attaining toward the end of a week the size of a pigeon's egg, hard and mobile. In a few animals transitory evening hyperthermia was noted. There was no tendency to suppuration, and the lesion gradually diminished, but some traces might persist several years. These animals reacted definitely to tuberculin from about the twentieth day on, but this reaction usually diminished and disappeared within a year. These animals exhibited remarkable resistance to reinfection, standing a dose of 5 mgm. of virulent bacilli without ill effects, although controls died of acute miliary disease of the lungs in thirty to fifty days. They survived contact with disease over eighteen months without any ill effects, but virulent bacilli did enter their lymph nodes and could be detected by inoculation in the guinea pig. Protective inoculation of young cattle, based on these experiments, was begun in 1921. The technique is as follows: A 10-cc. syringe with quite a long needle is filled directly from the vaccine ampule and the needle is thrust deeply into the connective tissue of the dewlap, toward the upper third of the skin fold. No special after care is needed, but they should not be submitted to tuberculin tests for quite a period because of their interference with the development of immunity. The appearance of resistance can be determined by the intravenous injection of 5 mgm. of virulent bacilli. It begins to appear about the fifteenth day or toward the middle of the febrile period, and is fully established by the twentieth day, lasting a year or more (fifteen months probably). A revaccination reëstablishes or prolongs it. A large rural farm in one of the Western Departments of France was available for practical testing of the method. This vaccination was systematically performed on all the calves as soon as possible after birth, other conditions and methods of bringing them up being kept the same. It had been customary to separate the mother from the calves soon after birth because of the epizoötic diseases which frequently attacked the calves. However they were fed on raw milk much of which came from tuberculous cows. In the course of six years, up to January, 1927, 70 calves, of whom 20 were from tuberculous mothers, were thus vaccinated and revaccinated. On this date 30 of the animals were slaughtered and carefully necropsied. No lesions were discovered in any of the vaccinated, which shows the incapacity of *BCG* to recover its original virulence after a sojourn of several years in living animals. These findings combat the view that *BCG.* should be reserved for children born of tuberculous mothers or brought up in a tuberculous environment. If *BCG.* is incapable of recovering its virulence in the bovine body, how much less likely that it should do so in the human body, for which it is infinitely less formidable! From September, 1924, to December, 1928, there were carried out, in France, Belgium, England, Holland and Italy, 20,358 bovovaccinations or revaccinations without incident. *Human Problem:* In the human race 93 to 96 per cent of adults, at least in large cities, react to the tuberculin test, indicating infection with the tubercle bacillus. In many cases such infections date from very early in life, and Calmette has traced their frequently occult course. Unfortunately these occult contaminations, while compatible with the appearances of health, seem to profoundly and definitely affect the organism; hence the value of *BCG.* to protect human beings as well as cattle. In July, 1921, Weill-Hallé first vaccinated a young infant in the maternity ward of la Charite. A few days after birth its mother had died of phthisis and it was to be brought up by its grandmother, also phthisical. The child has survived without any untoward illness. After thoroughly satisfying himself that *BCG.* was innocuous, by experiments on various animals, including anthropoid apes, Weill-Hallé resumed vaccinations of children and by January, 1924, had so treated 317 newborn, together with Turpin and Mlle. Coloni, with only one death at 4 months of a child suckled by its phthisical mother until her death in the hospital. Except 14 others, who died of nontuberculous maladies, all were perfectly developed and up to normal weight. Considering these results proof of the innocuousness of *BCG.* for the nursling, as well as for various animals susceptible to tuberculosis the Pasteur Institute decided to supply it to the general profession of France and to

foreign laboratories. Thus, from July, 1924, to July, 1928, data have accumulated rapidly. In France about 116,000 were vaccinated. Those born of tuberculous mothers or living in a tuberculous environment numbered 3,607. Elaborate inquiries have been made to learn the fate and state of health of those vaccinated. The known tuberculous mortality for the first year among the 3,607 last mentioned, including deaths from "meningitis," was 1.5 per cent, and from 1 to 4 years 0.3 per cent, while the general mortality (from all causes) has been respectively 7.3 per cent and 1.7 per cent. As there do not exist in France official figures covering these same ages there have been taken as controls children born of tuberculous mothers or born and brought up in a tuberculous environment, not vaccinated, but under the surveillance of the Ministry of Health through their dispensaries or offices. Replies were received from 204 of these, bearing on 4,854 unvaccinated children, of whom 1,989 were born of tuberculous mothers and 2,865 born or brought up in a tuberculous environment. Among the first group there was an 18 per cent mortality from tuberculosis and 24.2 per cent from all causes; among the second, 13.9 per cent from tuberculosis and 18 per cent from all causes. The same antituberculosis offices or dispensaries were able to follow in the years 1925 to 1927, inclusive, 2,368 infants vaccinated with *BCG.*, of whom 879 were born of tuberculous mothers and 1,489 born or brought up in a tuberculous environment. The mortality among the first group was, from presumable tuberculosis, 3.6 per cent, and from all causes 16 per cent. Among the second group it was, from presumable tuberculosis, 2.6 per cent, and from all causes 8.9 per cent. Summing up, the total tuberculous mortality was, in the unvaccinated 15.9 per cent, and in the vaccinated 3.4 per cent; the general mortality was, in the unvaccinated, 21.1 per cent, and in the vaccinated 12.5 per cent. Thus, for infants of the same age, brought up under exactly comparable conditions, preventive vaccination by *BCG.* reduced the tuberculosis mortality nearly three-fourths and the general mortality nearly one-half. The vaccinated children show a better resistance to measles, grippe, bronchopneumonia and intestinal affections, possibly due to stimulation of the lymphatic system. A study was also made of children born of tuberculous mothers and not separated, vaccinated between July 1, 1924, and July 1, 1927 and who have lived at least a year in continuous contact since birth, their records being followed up to October, 1928, or until death. There were 814 such children. Among these the tuberculous mortality from one month to four years was 2.4 per cent, and the general, from all causes, 10.6 per cent. During the same period the 204 antituberculosis dispensaries or offices reported 1,989 children born of tuberculous mothers and not separated or vaccinated. They showed a tuberculosis mortality of 18 per cent. Thus the protective value of *BCG.* for this class of children is conclusively demonstrated. Its innocuousness cannot be doubted. But nothing prevents its being renewed, for instance, at the end of the first and third years, and again at about ages of 7 and 15, for those who fail to react to the tuberculin test, which shows that they have either lost their immunity or escaped infection because of vaccination. For revaccination, a single subcutaneous injection of 0.05 or 0.025 of a mgm. is sufficient and causes no untoward reaction.—*La vaccination antituberculeuse par le BCG. Son Histoire,—ses Qualités, Guérin, Rev. Belge d. l. Tuberc., March-April, 1929, ii, 65.*—(*A. P.*)

Immunization with BCG.—The facts here set forth are the result of studies pursued from 1922 to 1928 with the collaboration of Benda and Mlle. Maas, physicians, and four visiting nurses, also named. Weill-Hallé himself had in 1921, eight months previously, made the first clinical trial of the vaccine. To maintain permanent medical surveillance over healthy children is not an easy task. Fixed consultations were, however, established, supplemented by domiciliary control by the visiting nurses. Observations made included weight, cutireaction, and other individual records. Some of the results have been published periodically at various stages. Since 1924 weekly consultations have been held at the *Hôpital Herold* and the *École de Puériculture* of the Faculty, and two supplementary ones each month at Belleville and Asnières. In addition, nearly 2,000 letters were sent out in 1927 and 1928. At each consultation about 16 children were examined, this being done every three months when possible, clinical examination being finally completed by cutaneous tuberculin tests, roentgenography and laboratory studies as indicated. The results assembled comprise the figures of morbidity and mortality among the vaccinated, compared with those established for the unvaccinated under similar conditions. There is a certain element of error due to the presence of a group which could not be controlled, amounting to 78 out of 613 by March, 1928. Since then the number vaccinated by ingestion has increased to 664, while the proportion of unknown has diminished. There remain 586 children followed up, among whom there were 42 deaths over

six years, or 6.32 per cent of the total 664, or 7 per cent of those followed up. We must now study separately those brought up in tuberculous and non-tuberculous environment. *Children Brought Up in a Tuberculous Environment:* There were 152 such, issue of tuberculous parents, and some of their histories are cited, which include quite a number of curable tuberculous incidents. The total number of deaths was 20, among which 95 per cent occurred during the first year. The percentage of positive tuberculin cutireactions increased steadily from 14.7 per cent at three months to 66.6 per cent at two and one-half years, and then diminished slightly. The percentage of deaths from sure or possible tuberculosis was 3.9 per cent during the first year, from controlled or positively determined tuberculosis 0.65 per cent. After the first year no deaths were recorded among vaccinated infants in a tuberculous environment (since 1922) excepting a single case at twenty months. These children may again be divided into three subgroups: (1) those born of tuberculous mothers, with or without bacillary sputum, (2) those born of healthy mothers but otherwise exposed to bacillary environment, and (3) those born of healthy mothers, exposed to other contacts, without determination of bacillary sputum. Tuberculin cutireactions were negative in the majority in each group. *Children Reared in Healthy Surroundings:* These included 392, vaccinated by ingestion of *BCG* and examined regularly thereafter. Among the maladies occurring were the ordinary toxiïnfections; also there were certain episodes of a tuberculous nature, the source of which was difficult to determine. *Tuberculin Cutireactions:* The percentage of positives increased from 4.5 per cent at three months to 14.2 per cent at the end of a year, 21.6 per cent after eighteen months, 23.3 per cent at two years, and 45.4 per cent at four years. Calmette argues that the development and persistence of cellular sensitiveness to tuberculin is ordinarily associated with follicular lesions, which, however, are not produced by *BCG*. Nevertheless *BCG* may set up ephemeral lesions, coinciding with a transitory sensitiveness to tuberculin. Its absence is no indication of lack of antituberculous immunity, however, since this state is independent of tuberculin sensitiveness. Thus are explained the possible variations of the cutireaction in the vaccinated. *Fatal Pathological Episodes:* There were 22 deaths among these 392 children (5.6 per cent), of which 17 occurred during the first year, 3 the second, and 2 the third. Most of these were due to ordinary toxiïnfectious causes but certain of them may have been of tuberculous causation. The presumably tuberculous deaths amount to 1.02 per cent, being highest the first year. *Revaccination:* Experimental studies have shown that vaccinal protection flags at about eighteen months and that one or more revaccinations are advisable. However, certain children vaccinated in 1922 have lived to date in tuberculous surroundings without their immunization being renewed. The anthropoid and pithecian apes vaccinated by Wilbert in 1923 still resisted (in 1927) contamination by tuberculous apes living with them. Still a revaccination at the end of the first and third year seems a wise precaution, performed when the child is enjoying good health. Over 200 such have been carried out, mostly with the cutireaction negative. *Vaccination by Injection:* Since 1924 the innocuousness of the vaccine seems clearly demonstrated. Frequently infants are seen who for various reasons were unable to benefit from early vaccinal ingestion. Also, the uncertain and slow absorption by this route makes a better method desirable. Subcutaneous injection has the double advantage of clearly demonstrating the innocuousness of the vaccine and of being applicable also to larger children, perhaps even adults. However, it is highly important to first determine that the organism is a virgin one as regards tuberculous infection and proof of non-contamination must be demanded. Otherwise we risk provoking Koch's phenomenon, a focal reaction, or generalization. Various authors have since pursued this method, notably Parisot, at Nancy, Colonial physicians such as Rousseau and Grofilez, Heimbeck in Norway, Wallgren in Sweden. The dose arrived at in the beginning was 1 to 2 mgm. containing 40 to 70 million bacilli. Following this, subcutaneous nodosities were observed, veritable tuberculous gummas, which ultimately broke down and discharged. To avoid these local lesions the highest dose was sought which would not cause abscess. Little by little the initial dose was reduced to $\frac{1}{4}$, then $\frac{1}{10}$, finally $\frac{1}{20}$ mgm. The injection is practised at the level of the axillary border of the scapular region, which is very accessible to the operator but not to the child and not very painful to puncture. Doses above $\frac{1}{20}$ mgm. almost invariably cause small abscesses, while less than $\frac{1}{50}$ mgm. rarely do so. There is a latent period of two weeks; then an indurated nodule is palpable under the skin and attains its maximum at five or six weeks; it then softens progressively while the skin becomes red and toward the end of the second month it "points" externally, discharging a thick creamy liquid, containing necrotic cells, polynuclears and macrophages,

with rare bacilli or granulations. In a few days it becomes more serous and at the end of a few weeks cicatrizes in a punctiform manner, some redness remaining for several months. With doses between 0.02 and 0.01 mg. the infiltration resolves without opening externally. By the fourth or fifth week the Pirquet reaction becomes positive, sooner than is the case with the ingestion method. Although Calmette has shown that allergy and immunity are not necessarily parallel, such early appearance of a positive cutireaction suggests associated biological changes leading to immunity. The subjects so vaccinated may be divided into two categories: (1) those definitely separated since birth or several weeks of age from contagious surroundings, and (2) those in the vicinity of a bacillary spitter or only recently separated. In the first case, two or more cutireactions, at weekly intervals, are carried out; in the second a month's observation is deemed necessary, when if the tuberculin cutireaction is still negative vaccination is performed. From April, 1924, up to date (1928) this method has been used on 35 infants, a very limited number compared with the 600 and more vaccinated by ingestion. There have been 6 deaths among these, one of bronchopneumonia complicating measles, 2 of convulsions suggesting meningitis, 2 of alimentary toxiinfections, and the sixth of bronchopneumonia. The health of the others appears satisfactory, although mostly living in a tuberculous environment. In most cases the tuberculin cutireaction has become positive since vaccination. *Conclusions:* The innocuousness of *BCG* vaccine has been clearly shown and its effectiveness proved. Seven consecutive years of study confirm this opinion. The objections put forth are theoretical. Vaccinal ingestion does not affect growth or development, even in the case of premature children. The clinical histories and experimental facts do not support the objection of possible return of virulence. Thus: *1:* Antituberculosis vaccination by *BCG* of previously uncontaminated subjects is quite inoffensive. *2:* It may be carried out either perenterally or subcutaneously. *3:* By the digestive tract the newborn receive 3 doses of 0.01 gm. *BCG* renewed at one and three years. *4:* By the subcutaneous route, 0.01 to 0.02 mgm. are given, after a period of observation with at least 2 negative cutireactions at weekly intervals. *5:* Isolation for a sufficient period following administration of the vaccine is recommended. *6:* The results obtained during over six years' observation, with confirmatory tests in France and foreign countries, warrant extending the method to threatened infants and those not threatened whose families ask for it, to adults, especially hospital attendants, exposed permanently to contamination, as yet free from infection, and to indigents from infected regions.—*Sur la vaccination antituberculeuse du nourrisson par le BCG (1921–1928), B. Weill-Hallé and R. Turpin, Rev. d. l. Tuberc., February, 1929, x, 9.*—(*A. P.*)

Use of BCG in Belgium.—In Belgium l'Institute Bacteriologique of the University of Liege was authorized to prepare and distribute Calmette's vaccine. In 1924 this service began to function for physicians in both Belgium and Luxembourg. By August 1, 1928, 1,111 children had been vaccinated with *BCG*, and demands for it came from 201 physicians, 14 maternity hospitals and the dispensaries affiliated with the *Ligue Nationale Belge contre la Tuberculose.* In 1929 information was requested of the physicians as to the state of health of 506 children vaccinated from one to four years,—from August 1, 1924, to August 1, 1928. Two hundred and eighty-three sufficiently definite replies were received concerning children regularly followed by the physician. Among those vaccinated one to two years 55 had been brought up in contact with tuberclebacillus carriers, 13 in contact with nonbacillary tuberculous persons, and 20 in a nontuberculous environment. Among those vaccinated two to three years 63 had been brought up in contact with bacillary tuberculous persons, 21 with nonbacillary tuberculous persons, and 38 in a nontuberculous environment. Among those vaccinated three to four years before, 43 had been brought up in contact with bacillary tuberculous, 10 with nonbacillary tuberculous, and 20 in a nontuberculous environment. The mortality from presumably tuberculous affections among all those in contact with bacillary tuberculous persons was less than 2 per cent, and the nontuberculous mortality 8 per cent. The latter included such causes as athrepsia, congenital debility, "convulsions," gastroenteritis, pneumonia and the exanthematous infections. These results may be compared with the results of investigations made by Falloise among the families of bacillary tuberculous persons for the League, finding a 20 per cent mortality among contact infants under a year, and with the still higher mortality of 29 per cent gathered by the dispensaries for the Arrondissement of Liége.—*Le dispensaire antituberculeux, J. Van Beneden, Rev. Belge d. l. Tuberc., March-April, 1929, xx, 77.*—(*A. P.*)

BCG.—Calmette answers some objections raised by Uhlenhuth and others concerning

the technique of application and the results from the use of *BCG.—A. Calmette and C. Guérin, Deutsche med. Wchnschr., December 27, 1929, lv, 2173.—(H. S. W.)*

Bacteriology of Pus from BCG Abscesses.—It is well known that subcutaneous protection by *BCG* vaccine gives rise, in about 10 per cent of cases, according to statistics from the Villemin Dispensary, at Nancy, to small local lesional reactions described by Weill-Hallé and Turpin, consisting in the appearance, at the end of a variable number of days, of a small subcutaneous nodule the size of a pea at the site of injection. These may develop in two ways: (1) retrogress or disappear without trace, or (2) increase, soften, fluctuate, and finally become true cold abscesses, curing quickly and definitely after puncture and evacuation. This pus, aseptically withdrawn, has been the object of investigation. Despite the small dose of vaccine (about 0.01 mgm. or about 400,000 bacilli) it was thought that if *BCG* were recovered, some new experimental knowledge could be gathered as to its stability and capacity for transmutation. In most instances 1.5 to 2 cc. of greenish pus were obtained and cultivated on ordinary gelatin and ascitic gelatin. The remainder was injected subcutaneously in the thigh of a guinea pig, each animal receiving a minimum of 1.5 cc. After a week's incubation, in all but one case the culture tubes remained sterile; this one showed a white staphylococcus, probably an accidental contamination. Microscopic examination showed acid-fast bacilli in only two cases, but if more material had been used and homogenized doubtless there would have been more. We can, however, state that the inoculated animals received these same acid-alcohol-fast bacilli and no others; hence their reactions would show if they were typical *BCG*, avirulent and incapable of tuberculization. None of the guinea pigs exhibited any symptoms and, in short, lived the same life as the controls. None of the necropsies showed any lesion of tuberculous appearance. As to the thorax, there were no tubercles, pulmonary congestion or abnormal intertracheobronchial nodes. Smears from the central parts of these nodes failed to show any acid-fast bacilli. *Conclusions: 1:* Subcutaneous injection of 0.01 mgm. of *BCG* may determine (in about 5 per cent of cases) a local reaction with the eventual formation of a small abscess which heals rapidly if punctured and evacuated. *2:* By direct examination of the pus one may find acid-alcohol-fast bacilli but no other organisms. *3:* Finally, these bacilli when reinjected into animals susceptible to Koch's bacilli are incapable of causing the development of tuberculous lesions in them: hence they remain avirulent *BCG*. It is therefore concluded that after two to four months' habitation in a human organism the *BCG* bacillus was incapable of recovering tuberculogenic properties and that the purely local incidents which may occur have nothing to do with true tuberculous lesions.—*Le pus des abscès consécutifs aux injections vaccinantes de BCG: son étude bactériologique et expérimentale, J. Parisot, L. Fernier and H. Saleur, Rev. d. l. Tuberc., February, 1929, x, 50.—(A. P.)*

Infantile Mortality in Tuberculous Environment.—It seemed desirable to compare the ordinary mortality under such conditions with that of infants vaccinated with *BCG*. First the mortality prior to 1921 (before the period of prophylactic organization) is compared with that of 1924–1928. The group selected comprises in part the old town of Nancy, the suburban villages, the industrial centre of Pompey Frouard, and that of Joeuf, to the Northeast, in the midst of a populous metallurgical basin. This is all included in the Department Meurthe-et-Moselle. In the period of observation indicated (July 1, 1924—December 31, 1927) there were 155 births. The children, all strictly supervised, have been in increasing proportion vaccinated with *BCG*. By July 1, 1928, there had occurred 32 deaths, of which 17 took place the first year, from all causes, or 10.96 per cent. If to these 155 births are added 221 children less than four years old July 1, 1924, we have a total of 376, of which during the period mentioned (up to December 31, 1927) 45 died between birth and four years, or 11.96 per cent. Among 55 new-born children receiving *BCG* there were 7 deaths, 1 attributed to tuberculosis, while among the nonvaccinated there were 14 deaths, 7 attributed to tuberculosis (or half the total). The following further statistics are cited: *1:* The 21 villages had at the census of 1926 a total of 51,802 inhabitants, and registered during the 1924–1928 period 4,546 births "domiciliated," of which 443 died in their first year during the same period, or 9.74 per cent. The town of Nancy, during the same period, had a mean mortality of 11.3 per cent. Its population in 1926 was 108,439. The infantile mortality recorded prior to 1921 during the first year in a bacillary environment was considerable, as Calmette found: 32.6 per cent for Paris, and 40, 60, and even 70 per cent for certain other districts or countries. For Meurthe-et-Moselle the mean figure was 18 per cent but varied greatly with hygienic and social conditions. The maximum figures were just short of 26 per cent. *Summary*

and Conclusions: The tuberculosis mortality which formerly was so formidable for young children in a bacillary environment has, through the concerted development of anti-tuberculosis prophylaxis, notably retrogressed. In Meurthe-et-Moselle it has in a few years fallen from 18 to 10 per cent. In addition to direct prophylactic measures must be considered such indirect ones as the reduction of alcoholism and syphilis, improved alimentation and upbringing, etc. These are as important in relation to the tuberculous child as to others—*Mortalité infantile dans les milieux familiaux bacillaires, J. Parisot and Saleur, Rev. d. l. Tuberc., February, 1929, x, 45.*—(*A. P.*)

Haemorrhagic Diathesis in Tuberculosis.—It is a commonly accepted fact among clinicians that certain individuals suffering from tuberculosis show marked tendency to haemoptysis, while others with lesions pathologically similar do not bleed at any time during the course of the disease. It has been found that those showing haemorrhagic propensities are often the victims of some latent blood dyscrasia. The findings are especially noteworthy in patients with copious and repeated haemorrhages, out of all proportion to the extent of the lesion. Fifty such individuals were chosen and studies were made to determine whether or not some blood dysfunction was present. The blood platelet count was below 200,000 in 29 cases; above 250,000 in 14 cases; between 200,000 and 250,000 in 7 cases. The bleeding time was longer than 4 minutes in 12 cases. The coagulation time was normal in all cases but one, in which it was slightly increased. Retraction of the clot was poor in 22 cases; fair in 12 cases; good in 15 cases; excellent in one case. Hess's test was positive in 10 cases. Koch's test was positive in 5 cases. The hammer test (gentle percussion of the skin over superficially placed bones and noting appearance of subcutaneous haemorrhage) was positive in 2 cases. A haemorrhagic diathesis is often a manifestation of lowered resistance and is commonly observed in many of the acute infectious diseases.—*L'emogenia acquista tubercolare, A. Rabino, Tubercolosi, March, 1929, xxi, 105.*—(*U. E. Z.*)

Cellular Reactions in Tuberculosis.—In a previous experiment large mononuclear cells and polymorphonuclear leucocytes containing tubercle bacilli were observed within the peritoneal exudate in guinea pigs infected with tuberculosis. The question arose whether the mononuclear cells were actually the monocytes of Sabin and whether the phagocytosis of the tubercle bacilli by these cells and by the polymorphonuclear leucocytes necessarily involved only the dispersal of the organisms throughout the animal economy, or whether this phagocytosis might not under some circumstances be a contributory cause to the destruction and elimination of the infecting bacteria. In fact, the possibility that under certain conditions phagocytosis by the polymorphonuclear cells in particular might be the chief agency in decreasing the number of viable organisms could not be lightly dismissed. Consequently experiments were undertaken to disclose certain facts with reference to the occurrence and activity of these cells in animals infected with tuberculosis. As the allergic state determines in a large measure the course of infection with tuberculosis, the cellular reactions of the peritoneal exudate following intraperitoneal injections of virulent tubercle bacilli were determined in both allergic and nonreacting animals. A series of 18 matched guinea pigs were divided into three groups. One of the groups received subcutaneously 1 cc. of a fairly heavy suspension of tubercle bacilli of low grade virulence (RI). Approximately three months later 4 guinea pigs of this group, now allergic (2 having died of intercurrent infection) and the 6 guinea pigs of another normal group were inoculated intraperitoneally with a suspension of virulent tubercle bacilli (culture no. 571). Prior to the inoculation, examinations of the peritoneal exudate in all the animals were made, and these examinations were continued under standard conditions at suitable intervals following the inoculation. Both the vital-staining technique and smears stained by the Wright method were employed. In the results recorded in the charts, values representing composite counts of the two methods are given. The changes in the nonallergic animal were as follows: Polymorphonuclear leucocytosis, monocytosis, a variable lymphocytosis with a persistent, though variable, monocytosis; and in the allergic animal: Polymorphonuclear leucocytosis, monocytosis accompanied by lymphocytosis, monocytosis, overshadowed by lymphocytosis. The response of the allergic animal differs from that of the nonallergic not so much in the quality of the cell response as in the quantity of cells to make their appearance and in the rapidity with which the reaction progresses throughout several stages. The rise in the number of the polymorphonuclear leucocytes and the lymphocytes is more abrupt and higher in the former than in the latter animal. In the case of the monocytes, however, although the initial rise is more rapid in the allergic animal, the level finally attained is generally higher in the nonallergic. In the above ex-

periment no observations were made prior to twelve hours after the infecting dose had been given. Two additional animals were taken, one allergic and the other nonallergic, and each was inoculated intraperitoneally with a fairly heavy suspension of R1 tubercle bacilli, and the peritoneal exudate was withdrawn at varying intervals beginning ten minutes after injection. It was found that in the first hour none of the monocytes of the allergic animal ingested organisms, while in the nonallergic animal the bacilli were phagocytized during the period by both the monocytes and the polymorphonuclear leucocytes. However, in the allergic animal 80 per cent of the polymorphonuclear leucocytes at the end of the first hour and 100 per cent at the end of the second hour had ingested tubercle bacilli, while the corresponding figures for the nonallergic animal were 16 and 29 per cent respectively. To determine whether the phagocytosis by the polymorphonuclear leucocyte resulted in a destruction of the organisms, an allergic and a nonallergic animal, previously treated with aleuronat suspension intraperitoneally and having developed the characteristic peritoneal reaction with leucocytosis, were inoculated intraperitoneally with a suspension of R1 organisms. After two hours the animals were killed and the washings from the peritoneal cavity in various dilutions were seeded on Petroff's medium which contained no gentian violet. After four months' incubation, growth was apparent in the lower dilutions but only in the tubes seeded with exudate dilutions from the nonallergic animals. As only individual animals were used in the last two experiments the results are suggestive but not conclusive. Apparently the phagocytes from the normal animal are not as active in destroying bacilli as those from an allergic one. To determine whether variants of the tubercle bacillus would produce a different cell picture an organism isolated from a child was "dissociated" by the method of Petroff. Two distinct colony types were obtained, but on inoculation into normal and allergic guinea pigs the cellular reaction was found the same for both. An attempt to determine the difference in the avidity with which the bacilli of the two types of colony would be phagocyted was made by the Wright opsonin method. When normal serum was utilized the amount of phagocytosis of the organisms of the R type was less than that of the organisms of the S type and the reverse was true when the opsonizing sera were from tuberculous individuals. *Summary:* The response of the peritoneal exudate depends primarily upon the allergic state of the animal. The differences in such response lie in the quantitative relationship of the various cell types involved as well as in the rapidity of the sequence of these reactions. The appearance of the polymorphonuclear leucocytes is not merely mechanistic in nature. They do not collect at the site of the tuberculous process solely because tissue has been injured, but this type of cell has in tuberculosis as definite a function to perform as in any purely pyogenic disease. From the data presented the function seems associated with the destruction of, or the inhibition of the growth of, tubercle bacilli, certainly of the less virulent forms. The reactivity of the tuberculous animal to the less virulent forms of tubercle bacilli seems to be increased, while the reactivity to the virulent form remains unchanged.—*Some Cellular Reactions and Immunity in Tuberculosis, M. B. Sherwood, J. Immunol., December, 1929, xvii, 481.*—(*J. S. W.*)

Hilar Reactions and Pulmonary Tuberculosis.—Infection by the tubercle bacillus, whatever the portal of entry, always results in the tuberculization of the tracheobronchial lymph nodes. Adolescent or adult pulmonary tuberculosis is of hilar origin and is first manifested by a tuberculous hilar or perihilar inflammation. Following the tuberculous tracheobronchial periadenitis, retrograde subpleural or perivascular lymphatic extension occurs. The involvement of the lymphatic system gives rise to tuberculous lesions of the pleura, intraparenchymatous tuberculous lymphoid deposits and alveolar or bronchial tubercles. The localization of pulmonary tuberculosis in the apices is purely a clinical finding and merely indicates the period when auscultatory signs are present. Anatomically, pulmonary tuberculosis arises at the hilum and its spread begins at this point.—*Les réactions hilaires commes point de départ de la tuberculose pulmonaire, J. Caussimon, Prog. Méd., January 19, 1929, no. 3, 101.*—(*U. E. Z.*)

Paralytic Disease in Guinea Pigs Due to Tubercle Bacillus.—In a series of tuberculous guinea pigs this disease-complex made its appearance abruptly when the animals had been injected two months, and after new cases had developed for a period of about ten days it disappeared equally as abruptly. The most obvious possibility was that it was a tuberculous meningitis, as the symptoms bore a close resemblance to those of experimental tuberculous meningitis in rabbits and dogs. To rule out the possibility of superimposed infectious disease of the central nervous system not due to tubercle bacilli the disease was maintained for study by serial passage in guinea pigs. This was accomplished by the intracerebral injection of

emulsions of brain from animals showing the paralyses. Three possibilities besides tuberculosis had to be considered: (1) infection with the filterable virus causing Roemer's guinea pig paralysis; (2) infection with a low-grade herpes virus since the culture of tubercle bacilli used in the experiment had been recently isolated from human sputum; and (3) infection with some visible organism other than the tubercle bacillus. The general plan followed was to remove the brain under aseptic precautions from a paralyzed guinea pig and to inject 0.1 cc. of a 10 per cent emulsion of the brain into a trephined normal animal. The disease was maintained in this way through 9 successive groups of guinea pigs and the clinical picture remained in all essential details the same throughout. The incubation period varied from 8 to 27 days, shortening at each successive passage, but remarkably constant for the animals of each group. The first symptoms of the disease noticed were a listlessness, a roughened fur and some faulty coördination. The last soon became marked and was most noticeable in the hind-quarters. Other symptoms referable to the central nervous system that were frequently present were hyperaesthesia, a tendency to move in circles, marked tremor of the head, and a tendency to torticollis. These were followed very soon by a definite posterior paralysis, spastic at first but later flaccid. Except for the day of inoculation, the animal appeared bright and alert even when there was complete posterior paralysis. As a rule, after three to four days of paralysis, the animal suddenly became worse, and usually death ensued in twenty-four hours. No animal in the series that was inoculated with a fresh brain emulsion from either a "spontaneous" or an experimental case was immune to infection. In animals in which the condition had been induced by intracerebral injection of brain emulsion from a previous case a few macroscopic changes were consistently found at autopsy. On opening the calvarium an excess of cerebrospinal fluid was always encountered. This was usually slightly turbid. Nothing abnormal was evident in the brain itself and rarely the meninges were adherent. There were no gross lesions in the cord. Outside the central nervous system the spleen was the only organ affected. This was usually somewhat enlarged and studded with hyperplastic follicles. Microscopic examination of brain sections showed a great abundance of acid-fast bacilli in the thickened pia mater but none elsewhere. The condition was found to be a true meningitis, with some extension into the encephalon, but only along the line of the larger blood-vessels. The exudate consisted chiefly of large mononuclear cells. Necroses were lacking and giant cells and definite tubercle formation were not seen. In the first of the brain emulsions tubercle bacilli could easily be demonstrated by the Gabbet method but in material from later passages this was no longer possible. Cultures of the brain emulsion on Dorset's egg medium regularly yielded typical tubercle bacilli. Cultures in a modified Noguchi leptospira medium showed a pleomorphic gram-negative bacillus in the upper growth. This was non-acid-fast by the ordinary Ziehl-Neelsen technique, but when decolorized after the method of Gabbet showed some acid-fast forms. The granular sediment (lower growth) was entirely non-acid-fast. After three passages on this medium guinea pigs were inoculated intracerebrally, some with the upper and others with the lower growth from one of these cultures. All developed the typical posterior paralysis with its accompanying symptoms. Animals inoculated subcutaneously with the upper portion of the culture shortly developed discharging nodes at the site of the inoculation and in the caseous material occurred organisms similar to those grown in the Noguchi media. Autopsy of these animals two months later revealed the presence of lesions typically encountered in guinea-pig tuberculosis. The Noguchi-medium organism was obviously a tubercle bacillus despite its atypical staining reactions. Two rabbits were inoculated with brain emulsion to determine whether the authors were dealing with a mild form of herpes encephalitis, or with Roemer's virus, which is described as specific for guinea pigs. The animals developed paralytic symptoms similar to those encountered in the guinea pigs. There was nothing in their disease to suggest herpes encephalitis. Brain emulsions from these animals injected intracerebrally produced the disease in a second series of rabbits. Five Berkefeld-N filtrates of brain emulsions from experimentally infected guinea pigs failed to produce the disease when injected intracerebrally into 16 guinea pigs and 1 rabbit. *Summary:* The data presented above indicate the condition to be a true tuberculous meningitis produced by a strain of tubercle bacilli, possessing certain atypical staining properties when grown in a modified Noguchi medium. The organism failed to pass a Berkefeld filter.—*A Paralytic Disease of Guinea Pigs due to the Tubercle Bacillus, R. E. Shope and P. A. Lewis, J. Exper. Med., September 1, 1929, l, 365.*—(*J. S. W.*)

Cultural and Staining Reactions of Paralysis-Producing Bacillus.—The strain of tubercle bacillus producing paralysis in

guinea pigs was isolated from the sputum of a colored adult with chronic pulmonary tuberculosis. The sputum showed many acid-fast bacilli by the Gabbet method. It was inoculated into guinea pigs, and cultures on Dorset's egg medium were made from the spleens of several of these animals. A subculture of this medium was used for inoculating the series of animals referred to in the preceding paper. The culture in question was apparently of the human type and was acid-fast, except that after decolorization by 30 per cent nitric acid, followed by alcohol, counterstaining with Loeffler's methylene blue showed a few blue-staining rods. When cases of paralysis appeared in the series of guinea pigs inoculated with this strain, a series of transfers was done from animal to animal by direct intracerebral inoculation of brain emulsion, as previously described. In the first emulsions made, bacilli were easily demonstrated by the Gabbet method, but in material used for later passages this was no longer possible. Paraffin sections showed an abundance of acid-fast bacilli in the pia mater of the affected brains, but none elsewhere. Cultures of the brain emulsions on Dorset's egg and other media usually resulted in a typical growth of tubercle bacilli. The cultures of special interest, however, were made on a modified Noguchi leptospira medium. Ordinary nutrient dextrose-agar was diluted 1 + 8 with distilled water, boiled, cooled to 50°C., and 10 per cent of sterile guinea-pig serum was added. After thorough mixing the medium was immediately tubed in long narrow culture-tubes (200 x 13 mm.). Portions of the cerebral emulsions were planted on these tubes, using a long capillary pipette in such a way as to form a thin streak from the bottom of the tube to the top along one side near the glass, with a small extra deposit on the upper surface of the semisolid medium. These plants were incubated at 37.5°C. in a moist thermostat for several months. The majority of the tubes showed the gradual development of a faint cloud in the upper 10 mm. of the medium. This eventually became coarsely granular. It could be transferred to other tubes and developed the same appearance. Exceptional tubes showed a faint haze toward the bottom with a clear intermediate zone. Smears of the growth when fixed with methyl alcohol and stained with Loeffler's methylene blue, using heat to the point of steaming, showed rather definite, faintly staining, thin, small bacilli. These bacilli were always gram-negative. When the culture material was introduced subcutaneously into guinea pigs, lesions of the lymph nodes were produced, and autopsy of the animals two months later showed the presence of lesions typical of guinea-pig tuberculosis. This made it evident that the organism grown in the Noguchi medium was a tubercle bacillus. Further study of the staining qualities of the bacilli grown on the semisolid medium revealed the following points: If the heat-fixed preparations were stained with carbol-fuchsin in the usual manner and decolorized by acid and alcohol separately it was found that 30 per cent nitric acid and 20 per cent sulphuric acid decolorized many but not all of the bacilli. The slightest application of alcohol, however, after these strengths of mineral acid removed all the stain. Even though much weaker mineral acids were used—5 per cent sulphuric, 3 per cent of either nitric or hydrochloric—alcohol later removed all the stain. If the gram stain was employed according to the general plan of the Much method, with some modifications the bacilli retained the stain. The essential feature of this method is that the gentian violet is phenolized (2 per cent) and the stain when applied is heated. The iodin treatment is also prolonged and decolorization is done with equal parts of acetone and absolute alcohol. Under these conditions all of the bacilli retain the gram stain. It has become a dogma in America that if a bacterium, after staining with carbol-fuchsin, will not withstand decolorization by 30 per cent nitric acid, followed by alcohol, it is not a tubercle bacillus. In Germany a bacterium to qualify as a tubercle bacillus must resist decolorization with 3 per cent hydrochloric acid in 70 per cent alcohol. The culture meets other classical requirements to the extent that it produces disease of the characteristic sort in animals, that the bacilli in fixed tissues resist decolorization, and that the cultures on Dorset's egg medium present typical characteristics. We are concerned with a bacterium which is moderately acid-fast, not alcohol-fast at all, and in which the particular method of fixation influences the result in ways not previously recognized. From the above it is concluded that Much, and those who have followed him, are right in their contention that the tubercle bacillus may at times be positive to gram stain and demonstrable by means of it, yet negative to the Ziehl-Neelsen method rigidly applied.—*The Cultural and Staining Reactions of a Strain of the Tubercle Bacillus Producing Paralysis in Guinea Pigs, P. A. Lewis and R. E. Shope, J. Exper. Med., September 1, 1929, l, 371.—(J. S. W.)*

Fate of Tubercle Bacilli in Reinfected Rabbits.—In a previous study cultural methods were used to determine the fate of both human and bovine tubercle bacilli in the

organs of intravenously infected normal rabbits. In general it was shown that both types of bacilli at first multiply in all the organs, the human more rapidly than the bovine. Later the faster-growing human bacilli are more or less destroyed whereas the bovine bacilli, although ultimately destroyed in the liver, spleen and bone marrow, continue to grow in the lung and kidney without effective opposition until the death of the animal. Thus it would appear that as a result of the growth of both types of bacilli in the rabbit some change which destroys the bacilli takes place in the body. One would expect that after such a change has once been brought about the introduction of fresh tubercle bacilli from without would be followed by immediate destruction without any preliminary growth. To test this hypothesis, and to ascertain the fate of tubercle bacilli in the organs of reinfected rabbits, the following experiments were performed: A series of rabbits was inoculated intravenously with 0.001 mgm. of a human strain of tubercle bacilli per kg. of body weight. Six months later these rabbits were given intravenously a reinfecting dose of 0.01 mgm. of the same strain of human tubercle bacilli. Another series of rabbits was similarly infected with the human strain and reinfected with a bovine strain in the same quantities and by the same route. At the same time two series of normal rabbits were injected intravenously with 0.01 mgm. of the same strains used for reinfection, one with the human and the other with the bovine bacilli. At intervals of one day, one, two, four and six weeks and two months, equal amounts by weight of suspension of ground lung, liver, spleen, kidney and bone-marrow in varying dilutions were seeded on Dorset's and Petroff's media both directly and after sodium-hydroxide treatment. The number of colonies of tubercle bacilli appearing on each tube was repeatedly determined, the final readings being made after three months' incubation. The findings are reported for each organ in the form of tables in which the number of colonies both for the infected and the reinfected are arranged side by side for comparison. The general pathological findings for each animal are also noted. *Results: 1:* In the presence of a certain amount of residual primary lesions, human or bovine tubercle bacilli of reinfection were destroyed in all the organs of rabbits without any preliminary multiplication. This destruction was not quite complete; a few organisms persisted even two months after infection. *2:* No macroscopic tuberculous lesions due to reinfection developed in these rabbits. *3:* With practical disappearance of the primary lesions and their enclosed organisms, restricted multiplication of the bovine bacillus of reinfection took place but not the extensive growth of the virus observed in rabbits after a primary infection. *4:* In these rabbits slight tuberculous lesions developed as compared with the massive tuberculosis of primarily infected rabbits. *5:* The human tubercle bacillus of reinfection did not multiply at all, nor did it cause any lesions, even when the lesions of the primary infection had almost disappeared. *6:* Although the rabbit destroys efficiently considerable quantities of bovine tubercle bacilli of reinfection introduced from without it may harbor innumerable human bacilli in the residual primary lesions of the lung and kidney.—*The Fate of Tubercle Bacilli in the Organs of Reinfected Rabbits, M. B. Lurie, J. Exper. Med., December 1, 1929, l, 747.—(J. S. W.)*

Tubercle Bacilli in Faeces.—Unless tubercle bacilli are found, a positive diagnosis of tuberculosis cannot be made. In patients with sputum negative for tubercle bacilli, examination of the blood for their presence is often of no avail. Some observers have found tubercle bacilli in the faeces of all patients presenting a positive sputum. In patients whose pulmonary lesions have undergone retrograde changes and who no longer cough or expectorate, examination of the faeces offers another means of verifying the quiescence of the lesion. This method is of greatest value in small children and infants who cannot expectorate. It is also of value in patients who will not cough for legal or other reasons.—*Importanza della ricerca del bacillo di Koch nelle feci, M. Mazzucato, Tubercolosi, xviii, April 1928, 135.—(U. E. Z.)*

Tuberculin Type of Sensitization with Egg-White.—In certain previous studies the author has shown that guinea pigs infected with tuberculosis and treated with various protein substances (egg-white, horse-serum, timothy pollen, etc.) develop a skin sensitiveness similar to the tuberculin sensitiveness. In the present paper several methods are described for producing a tuberculin type of sensitiveness with egg-white in tuberculous guinea pigs. The different methods have this in common, that an extensive local lesion is produced, usually with pronounced inflammatory reaction, and the egg-white is introduced into the lesions. The injection of killed tubercle bacilli into the tuberculous lesions previous to the egg-white sensitizing injection is a favorite method. The guinea pigs are infected in both groins with moderate amounts of virulent tubercle bacilli. When the local lesions begin to swell these are injected twice with killed

tubercle bacilli in saline suspension. In this way a very hard swelling is obtained, with more or less inflammatory reaction in the surrounding tissues. Three or four days later 0.1 cc. of a 1:50 egg-white dilution, containing 0.5 mgm. of killed tubercle bacilli, is injected first in one lesion and the next day in the other. The results were no better with living than with killed bacteria. The subcutaneous injection of the egg-white did not sensitize. When using an avirulent strain of tubercle bacilli (R1) the guinea pigs are injected twice in the groins with a rather heavy emulsion. When a hard swelling appears this is injected with 0.1 cc. of a 1:50 dilution of egg-white without the addition of killed tubercle bacilli. The guinea pigs thus treated will even give the tuberculin type of sensitiveness when the egg-white is injected intravenously. When it is necessary to use killed tubercle bacilli for the preparation of the animals, this may be done by first injecting the dead bacteria intraperitoneally and a few days later the same dose of bacteria with the addition of a small dose of egg-white, or with a large dose of coagulated and finely ground egg-white. In guinea pigs treated in this way the omentum is transformed to a large tumor, adherent to the peritoneum and intestines and containing tissue similar to a true tuberculosis lesion. The killed-tubercle-bacillus emulsion, used for injecting the ground nodes, is prepared by immersing thin tubes of the emulsion in boiling water for two minutes. To inject egg-white into the lesion a small hole is cut through the skin with a pointed knife. This has the advantage that it is possible to see that the injection is made into the lesion, and if some of the injected fluid comes back, it can be wiped off and does not diffuse into the subcutaneous tissue. White guinea pigs, preferably males, weighing at least 450 gm., and usually more than 500 gm., were used.—*The Technic of Producing the Tuberculin Type of Sensitization with Egg White in Tuberculous Guinea Pigs, L. Dienes, J. Immunol., December, 1929, xvii, 531.*—(*J. S. W.*)

Anaphylaxis with Carbohydrate Antigen of Tubercle Bacillus.—In 1927, Tomcsik, working with the carbohydrate (the so called specific soluble substance or partial antigen) of *B. lactis aerogenes*, showed that guinea pigs passively sensitized with homologous antiserum died acutely when injected with the specific soluble substance. These observations were extended by Tomcsik and Kurotchkin to include the carbohydrate obtained from the pneumobacillus and yeast. In 1929 Avery and Tillet produced with the specific carbohydrate of the pneumococcus anaphylactic shock in guinea pigs treated with antipneumococcus rabbit sera corresponding to types I, II and III. The type specificity of the carbohydrate held rigidly in its anaphylactic rôle. It thus appears probable that in every case, in which a specific carbohydrate can be obtained from a complete antigen complex capable of producing antibodies *in vivo*, anaphylactic shock can be elicited under suitable conditions by means of the partial antigen. The experiments reported in the present paper were designed to confirm this hypothesis with regard to the carbohydrate of the tubercle bacillus. The specific carbohydrate used in this work was derived according to the process of Mueller from O. T. produced by a human strain (H37 Saranac) grown on a synthetic medium. It does not give a skin test in tuberculous animals or animals rendered hypersensitive to tuberculin by injection of killed tubercle bacilli. It gives a ring test with the homologous antiserum in a dilution of 1:2,000,000. Two milligrams of this substance invariably produced anaphylactic shock, often lethal, in guinea pigs passively sensitized with serum from rabbits treated with dead tubercle bacilli. The same shocking dose of the carbohydrate substance also produced acute lethal shock in guinea pigs actively sensitized with heat-killed tubercle bacilli. The uteri of guinea pigs, both actively and passively sensitized, gave typical reactions when the carbohydrate substance was added to the bath. The highest dilution employed gave a reaction in 1:100,000. Simultaneously with the determination of these facts it was possible to observe the effect of anaphylactic desensitization on the allergic reaction in those animals which suffered acute sublethal shock. By injections of 0.1 cc. of 10 per cent O. T. intracutaneously, before and after the shocking dose was given, any diminution of the skin lesion could be nicely perceived. The acute shock was found not to influence the intensity of the cutaneous response as indicated by the subsequent test with O. T. None of the guinea pigs passively sensitized with antituberculosis rabbit serum showed a positive skin test when tested with O. T. This is an indication that the anaphylactic mechanism is totally distinct from the phenomenon of skin allergy. The question naturally arises whether the instances of fatal shock reported were caused by the specific carbohydrate itself or a protein impurity, and whether actual anaphylactic shock had been produced according to the criteria of Wells. Since the specific polysaccharide contains 0.3 per cent nitrogen, it would seem theoretically impossible to exclude the participation of a protein in the reaction, but

this is exceedingly unlikely if we consider the small amount of protein represented by the lethal dose of 2 mgm. of carbohydrate. If all the nitrogen present be taken as included in protein, the latter would weigh about 0.0375 mgm. According to Wells, the minimal lethal dose of purified protein is about one-twentieth or one-tenth of a milligram. The process of purification, however, as well as the failure to obtain any qualitative tests for protein make it highly improbable that the nitrogen present represents a whole protein or even any of its higher derivatives. It seems reasonably certain that the substance responsible for acute death is of the nature of a carbohydrate. All but one of Wells's criteria for true anaphylaxis have been met in these experiments. The criterion that passive transfer of sensitization be possible was done only with rabbit serum, not with the homologous serum of the sensitized animal. Nevertheless it seems fairly assured that true anaphylaxis can be produced by the specific carbohydrate of the tubercle bacillus.—*Anaphylactic Shock with the Partial Antigen of the Tubercle Bacillus, J. T. Enders, J. Exper. Med., December 1, 1929, l, 777.—(J. S. W.)*

Sensitiveness of Guinea Pigs to Tuberculin.—In a previous paper it was demonstrated that young guinea pigs infected with tubercle bacilli show slight if any skin reaction to a watery extract of tubercle bacilli. In the following experiments the toxicity of Old Tuberculin for young tuberculous guinea pigs was determined. One or two days after birth 27 guinea pigs were infected subcutaneously with 1 mgm. of virulent human tubercle bacilli. From twenty-five to thirty-one days later they were injected intraperitoneally with 0.3, 0.6 or 1.22 cc. of a 1:5 dilution of Old Tuberculin. The intracutaneous test was given to 15 guinea pigs with 0.1 cc. of tuberculin diluted 1:10. Similar experiments were made on 28 adult guinea pigs weighing from 400 to 450 gm. Of these 12 were given intracutaneous tests. The results showed that young tuberculous guinea pigs are sensitive to the toxic action of tuberculin and that the lethal dose is smaller (5 cc. of a 1:5 dilution, in comparison with 22 cc.). Of the 15 young tuberculous guinea pigs 9 did not react to the intracutaneous test; 4 reacted with slight swelling that disappeared in two days; 2 reacted with redness, swelling and oedema. All 12 of the adult guinea pigs reacted to tuberculin. The observed lack of sensitiveness of the skin of young infants to irritants, the work of Valtis, and the experiments reported here suggest that the mechanism of sensitiveness to tuberculin is completely developed in young guinea pigs, and that the dermal reaction is obscured by some physiological and anatomical condition of the skin in the young animal. But this conclusion cannot be drawn because it cannot be said positively, even of adult guinea pigs, that the same mechanism is operative in the systemic and in the skin reactions.—*The Sensitiveness of Tuberculous Guinea Pigs One Month Old to the Toxicity of Tuberculin. J. Freund, J. Immunol., November, 1929, xvii, 465.—(F. G. P.)*

Tuberculin Allergy and Antituberculosis Immunity.—In 1903 Pirquet gave the term allergy to the phenomena of altered reaction of the tuberculous to tuberculin and of the anaphylactic to serum. This was the first of a series of studies which resulted in the skin reaction. To give a positive reaction it is not necessary to have a clinical lesion, as a mere infection is sufficient. On the other hand, a nonreactor or a patient in a state of anergy is not necessarily free from a bacillary lesion. Some animals, such as different species of monkeys, are only slightly sensitive to tuberculin locally when they are infected with tuberculosis. At times young guinea pigs and young children may not react, so that it is evident that a subject can be infected by tubercle bacilli (or even be a carrier of a latent tuberculous lesion or on the way to curing a lesion) without presenting the least tuberculin allergy. Antituberculosis immunity has very particular characteristics. It is followed by a mixed state of hypersensitiveness or allergy, toward the specific proteins produced by this germ and an intolerance to reinfection. This intolerance to reinfection is what determines the "phenomenon of Koch" and is antituberculosis immunity. This immunity may be established long before the appearance of tuberculin allergy or it may disappear long after the latter has ceased to exist. Consequently, antituberculosis immunity and tuberculin allergy are two states, distinct and independent, of the organisms infected by the bacillus of Koch or vaccinated by the *BCG*. Immunity may be present without allergy. The experimental and clinical proofs for this statement are cited. During the first days of life the intestinal epithelial cells are entirely protoplasmic. Mucus does not appear in them until the end of the tenth to fifteenth day. That is why the permeability of the intestinal wall is so great for microbes and albuminoid substances (toxins or antitoxins), and this fact forms the basis for the prophylactic inoculation of *BCG* by mouth during the first days of life. It produces an immunity but there may be no allergy. The absence of tuberculin reactions has led many pediatricians to question this immunity. To

prove the presence of the latter the reduced mortality from tuberculosis in 150,000 vaccinated children in France alone is cited. Also the autopsies, carefully performed, with experimental control, on more than 200 children who, after being vaccinated with *BCG* routinely, have later died of diverse conditions, have shown not only total absence of suspicious tuberculous lesions, but also the appearance of acid-fast bacilli in the lymph nodes. These bacilli have been identified as *BCG*, and they have been shown to be deprived of all virulence for guinea pigs. It is probable that if vaccinated children were skin-tested every month or every two months, preferably by the intracutaneous method, most of them would give a positive reaction at a given time. This reaction may be only transitory. Infection by virulent bacilli results in true tubercle formation with caseous centres, whereas with *BCG* it does not pass the stage of giant cells, or *granulation grise*, which may be indispensable for the production of tuberculin allergy. *BCG* given in large single doses into a lymph node or hypodermically, creates immediately a follicular lesion, provokes the formation of giant cells (which are absorbed and disappear later), and soon causes to appear, at the same time as the products of cellular reaction (mixed cellulobacillary proteins and antibodies), tuberculin allergy. While antituberculosis immunity may exist with allergy it is true that the only evidence of bacillary infection is the presence of allergy. Because of this, it is desirable to have patients allergic, and therefore Calmette proposes to examine at the end of the first, fifth, seventh, fifteenth and twentieth years those patients who were given *BCG* by mouth. If the tuberculin skin reaction is not present at these times, he proposes to revive it or produce it by the subcutaneous injection of 20 mgm. of *BCG*. This small dose is tolerated without the least danger or reaction and it is sufficient to provoke or reproduce tuberculin allergy and will therefore indicate the presence of tuberculin immunity. This work is, of course, still in the experimental stage, and only time can positively affirm or deny it.—*Allergie tuberculinique et immunite anti-tuberculeuse, A. Calmette, Ann. d. Méd., April, 1929, xxv, 293.*—(*B. T. McM*).

Cholesterol Agglutination Reaction in Tuberculosis.—Serum reactions have been used to a varying extent in the diagnosis of tuberculosis for more than thirty years. Complement fixation was used to a moderate extent some ten years ago and modifications of the technique are still being worked with, especially those of Besredka, Wassermann, Stimson and Wadsworth. However its reliability has yet to be demonstrated by experience. Precipitin reactions have been described as far back as 1904, but the only approach to a practical application is, or may be, that of Doan, who used a lipoid extract. Agglutination of tubercle bacilli by specific antisera was first recorded by Arloing in 1898, later by Koch in 1901. No satisfactory clinical method has resulted. The Vernes resorcin reaction is nonspecific in character and positives occur with other diseases than tuberculosis; hence, though considerably used in France, its value relates rather to "activity" determination. About two years ago Hinton, while working with bacterial lipoids in syphilis, observed that the serum of tuberculous patients agglutinated particles of cholesterol suspended in a mixture of glycerol, hypertonic salt solution and the alcoholic extract of ether-insoluble residue of an unknown strain of dried tubercle bacilli, while the serum of nontuberculous individuals did not. On the basis of this observation he obtained three strains of tubercle bacilli, H37, R1 and *BCG*, from the Trudeau Sanatorium, the Metropolitan Life Insurance Company Sanatorium, the Pathological Department of the University of Chicago, and the Biological Laboratories of H. K. Mulford Company. These were grown on two synthetic liquid media, that of Sauton and of Long. After about two months' growth the media were filtered off, the bacilli washed with distilled water, and placed, moist, in an incubator at 56°C. for several days. They were then kept in glass-stoppered bottles about four months and then treated by the method of Neymann and Gager for preparing Wassermann antigens. This consists in separate ether extraction (8 volumes ether to 1 part by weight of bacilli) four times, with shaking for 10 minutes during each. Finally the ether-insoluble residue was extracted with 95 per cent alcohol, 5 volumes. This lasted for 3 days, with occasional vigorous shaking, at room temperature. After trying various proportions experimentally the proportion of one part of the alcoholic extract of the ether-insoluble residue to nine of a 0.7 per cent alcoholic solution of cholesterol was selected as giving the best results. One part of this mixture was added to two of a 5 per cent aqueous sodium chloride solution and shaken vigorously. Twelve more parts of the latter were then added and the whole shaken. Finally 15 parts of 50 per cent neutral redistilled glycerine were added and the whole quickly shaken. This mixture is referred to as a *glycerinated indicator*, of which only enough is prepared for each day's experiments. *Technique of Test:* Into 3 agglutination tubes are pipetted 0.1, 0.3, and 0.5 cc. of serum. (These tubes

are 10 mm. in diameter and 100 mm. in length.) To each is added 0.5 cc. of the glycerinated indicator described. They are then well shaken in the rack, incubated overnight at 37°C., and the results noted. Sera from nontuberculous individuals produce no change or at most a slight scummy meniscus. Sera from known tuberculous individuals produce a definite agglutination of the cholesterol particles into clumps, with clearing of the fluid. In weaker reactions the clearing and clumping are less marked. At times tuberculous sera react differently with extracts from the three strains. *Results:* Out of 18 sputum-positive cases at the Boston Tuberculosis Sanatorium the agglutination reaction was positive in 16 and doubtful in 2. Out of 107 presumably nontuberculous cases at the Boston Lying-In Hospital, 2 showed a positive agglutination. In one of these tuberculosis had previously been suspected and in any event 2 per cent is not altogether unexpected among presumably nontuberculous individuals. *Summary and Conclusions:* An accurate diagnosis of so called clinical tuberculosis requiring treatment is often extremely difficult. The availability of any laboratory method must ultimately rest on a careful clinical investigation which requires organized coöperation and this is difficult to effect. However, such an investigation is about to begin at one of the better organized Boston Hospitals. So far as known the method here described and used is quite novel.—*Cholesterol Agglutination Reaction in Tuberculosis, W. A. Hinton and G. O. Stuart, New England J. M., February 13, 1930, ccii, 327.*—(*A. P.*)

Calcium Exchange in Pulmonary Tuberculosis.—Of all mineral elements calcium has undergone the most investigation in tuberculosis and is still the subject of ardent polemics and numerous publications. The entire subject is here reviewed in a general way. *1: Decalcification:* Recent investigations carried out with scientific precision have modified previously accepted ideas on calcium exchange. Marcel Labbé last year devoted one of his pathological lectures to the rôle of calcium in tuberculosis and some of his passages are cited, especially his statement that "the dogma of decalcification, which has been accepted and ruled tuberculosis therapeusis for many years is to-day upset." He goes on to say that Boyer in 1869 mentioned decalcification of the lung in phthisis, but it is chiefly to Ferrier that latterly accepted theories are due. He did not bother to study the calcium balance; instead he simply placed the patients in a bath and noted how easily their bodies floated. Ferrier's other arguments had to do with the relation between dental caries and bony demineralization, the calcium reserve, and the distribution of tuberculosis as compared with the calcium content of the water. Most French authors contend that decalcification begins in the early stages of tuberculosis and disappears in advanced cases. They determine it simply by estimation of the degree of calcuria (Senator, Stokavis, Arloing, Dumarest, and others). Robin gives the normal urinary calcium as 0.281 gm. per day. *Criticism and Analysis of the Foregoing:* In the first place, to consider only the degree of calcuria is very misleading. This has been shown to vary from 0.16 to 0.69 gm. even on an identical regimen. In fact most of the calcium is ordinarily excreted in the faeces. The only really scientific method of estimation is to determine the calcium balance or relation between total intake and output. Lanfer and Lematte have done this in tuberculosis patients at rest and on a measured ration. They found a negative balance in certain tuberculosis patients in whom active disease caused wasting. In these a dose of 2 gm. calcium oxide per day was necessary to compensate for losses. Except for these conditions tuberculous persons behaved about normally and consumed daily about 1.5 gm. calcium oxide. Hence, they concluded that decalcification is not a specific characteristic of tuberculosis but is associated only with denutrition. Manoussakis even found positive balances in a number of tuberculous persons. *2: Frequency of Dental Caries in the Tuberculous:* Ferrier declared that dental decalcification went hand in hand with skeletal. Others have been unable to confirm this and Villemin pointed out that in rachitic children dental lesions were rare. *3: Are Tuberculous Tissues Decalcified?* Barkus was unable to determine any loss or any increase as cure occurred and in guinea-pig experimental tuberculosis could not demonstrate any diminution of calcium. Maver and Wells even found an increase in animals necropsied. Other workers have shown that the calcium content of organs varies with the physiological state. *4: Blood Calcium of the Tuberculous:* Here recent studies have given the *coup de grace* to old theories. Tuberculosis *per se* causes no lowering of blood calcium (Wells, Halverson, Sweany and others). Haemoptoic patients with prolonged coagulation time showed slightly lower concentrations but the differences were slight. The normal variations (P. Matz) are from 9.22 mgm. per 100 cc. serum to 10.78 mgm. Sergent and Binet have found from 120 to 130 mgm. per litre even in cavity and bronchopneumonic cases. Howk and Medlar conclude that the tuber-

culous blood is not impoverished in calcium. Moreover, variations when they exist are dependent on cachexia or alimentary insufficiency. *Recalcification:* Despite these objections the dogma has been reiterated that the great curative principle in tuberculosis is recalcification. This is carried out (1) by administering calcium phosphate and other salts, and (2) by instituting a dietary regimen. The only demonstrable advantage of calcium medication is that it may act favorably on the stomach, which often in the tuberculous shows hyperchlorhydria. Denis and Minot have shown that oral administration of calcium lactate does not increase the blood calcium while Maver and Wells have shown that a calcium-rich dietary will not increase tissue calcium. Furthermore, in the tuberculous, calcium chloride or lactate may even increase calcium loss, especially the chloride. The glycerophosphates and carbonates are not assimilated at all and even increase calcium loss (Manoussakis). Laufer has further shown that phosphorus and calcium have a certain optimum relation, realized in the tuberculous when there is a slight excess of calcium, for instance, 1.6 to 2 gm. If this is exceeded, there is a tendency to the formation of insoluble calcium phosphate, which is eliminated by the intestine,—therapeutic decalcification. Calcium therapy might be useful when there is insufficient alimentary calcium, as in Germany during the War, but metabolic disorders can not be combated, that is, the organism cannot be made to fix calcium. Wells has shown that superfluous quantities are rapidly precipitated in the tissues and that the blood is normally physiologically saturated. *Fixation of Calcium:* Sergent believed that the addition of adrenalin would solve the problem, and Colin and Richard reported diminished calcuria under this regimen. However, these results have already been questioned by Bernard and Vitry, while Manoussakis showed that there was always a corresponding intestinal debit. He proposed parathyroid opotherapy to favor assimilation. Others have used solar light, ultraviolet radiation, lipo-soluble vitamine A, but none of these methods is effective. In rickets only the ultraviolet rays and codliver oil, containing vitamine A, protected animals, and this condition differs essentially from tuberculosis. *Calcification of Tubercles:* This has been considered manifest proof of the defensive rôle of calcium in "walling off bacilli." However, these salts are only deposited in necrotic tissue (Maver, Wells, Caldwell). Nothing proves that calcium precedes and favors cicatrization of lesions and Watt is not even convinced that their progress is arrested. It is much more probable that calcification follows rather than effects a cure. In bovine tuberculosis large quantities of calcium are deposited in and about progressive lesions (Küss, Pietttre). Also, bony tissue offers no special resistance to tuberculous infection. *Lime-Kiln Workers:* Individuals who breathe lime dust all day possess a special resistance to tuberculosis. This is incontestable. But its action, according to Wells, may well be a simple mechanical one, stimulating sclerosis. *Calcium in Experimental Tuberculosis:* Calcium salts do not inhibit the development of the tubercle bacillus; on the contrary, they often favor growth on culture media (Tanaka, Kindberg). Neither has any favorable effect been noted in tuberculous animals. *Conclusions:* If there is no decalcification, still the tuberculous need a little more lime than normal persons to maintain a favorable balance. Delore believes that calcium may stimulate phagocytosis and certain other favorable reactions. However, intensive calcium therapy, on the basis of the foregoing work, must be rejected as both useless and harmful. Nature furnishes abundant calcium, especially in water, milk, eggs and legumes. The best alimentary medium is milk, of which 0.5 litre per day, with leguminous purées or eggs, suffices. Articles which tend to precipitate calcium in the form of oxalates,—rhubarb, sorrel, cocoa, endive,—should be avoided. If in the presence of food rich in calcium it is not assimilated, the organism is at fault physiologically. This is often the case in tuberculosis, especially when oxidation is impaired, with a resultant tendency to humoral acidity. In such cases Loeper administers phosphoric acid, which indirectly favors retention of calcium. The biphosphates and lactophosphates can also be used.—*Les échanges calciques chez les tuberculeux pulmonaires, R. Monceaux, Rev. Belge d. l. Tuberc., March–April, 1929, xx, 49.*—(*A. P.*)

Bruit d'Airain.—This sound can be heard only when an air-containing cavity such as a pneumothorax, a pulmonary cavity, or, very rarely, a pneumopericardium is present. The term "coin sound" is misleading, because the media of percussion do not influence the *bruit d'airain*, whose genesis is inherent within the cavity. The presence of the sign is diagnostic of an air-containing cavity, but conversely the absence of the sign does not exclude the presence of such a cavity. The history of the development of the sign is summarized, and it is evident that opinions are not agreed as to the mechanism of its production. A study of 50 cases is reported. Of these 26 were males and 24 were females. Pneumothorax was induced

on the right in 28 cases, and on the left in 22. There were 2 cases of simultaneous bilateral pneumothorax. The study covered a period of two years. Each case was examined many times, in all stages of lung collapse, for the presence or absence of the *bruit d'airain* in relation to varying conditions, such as the development of pleural effusion, intrapleural tension, selective collapse, hydropneumothorax, mediastinal displacement, ballooning of the pleura, and changes in the walls of the pneumothorax cavity. At certain stages and under varying conditions in the same cases the sign was present at times and absent at other times. But, in the 50 cases, the sign was present at some stage over 28 pneumothoraces, and absent in all stages over the remaining 22. Of the 28 pneumothoraces exhibiting the sign, 23 developed pleural effusion, 3 gave a history or other evidence of fibrinous pleurisy, and 2 exhibited the sign only when there was evidence of mediastinal displacement or pleural ballooning. In 4 of the series the appearance of the sign where it had not been heard before was followed by the development of effusion. In the 24 pneumothoraces in which the sign was never found there was no incidence of pleurisy or effusion. The majority of these cases had good lung collapse, and in some the collapse was complete. In cases of hydropneumothorax the *bruit d'airain* was heard when the effusion was small or moderate, and became more intense as the effusion was absorbed. In each of the two cases of simultaneous bilateral artificial pneumothorax effusion developed on only one side and the *bruit d'airain* was heard only over that side subsequent to the development of the effusion. The amount of lung collapse was equal on both sides in the two cases. Intrapleural tension was not a factor which determined the occurrence of the *bruit d' airain*, but with positive pressure it was usually more intense. The presence of pleural adhesions seemed to influence the occurrence of the sign only in so far as their presence rendered the patient more liable to the development of effusion or obliterative pleurisy. The shape of the cavity was of minor importance, and its size or volume was of moderate importance in the production of the sign. A faint *bruit d'airain* could occasionally be heard in mediastinal displacement or pleural ballooning. All the metallic phenomena (*bruit d'airain*, amphoric breath-sounds, metallic vocal resonance, tinkling crepitations, and succession splash) were occasionally heard in the same case at the same time. In a dry pneumothorax with completely collapsed lung and hardened pleura the *bruit d'airain* was the only metallic phenomenon found. The sign was occasionally localized, depending primarily upon the location of the air space. The main point of the thesis is that in all cases in which rigidity of the cavity-wall was present, the *bruit d'airain* was heard. The *bruit d'airain* is of value in determining the extent of mediastinal displacement and of pleural ballooning, and in estimating the location of the fluid level in a hydropneumothorax or pyopneumothorax.—*Observations on the Bruit d'Airain (Coin Sound, Bell Sound), A. I. G. McLaughlin and A. J. Dix-Perkin, Lancet, December 28, 1929, ccxvii, 1351.*—(*R. M. F.*)

Pseudocavity Syndrome.—The stethoscopic findings of cavity, so accurately described by Laennec, were accepted by all the great clinicians of the nineteenth century, and not only the existence but also the extent of excavation was estimated by his method. With the advent of radiology greater precision was given to the diagnosis of cavity, and it was also conclusively demonstrated that in some instances signs of cavity may be present without corroborative evidence on the X-ray. In such cases there was invariably pulmonary sclerosis with deviation of the trachea. Even before the use of X-ray there was mention of a pseudocavity syndrome. In 1838 Williams pointed out a type of tympany that should not be confused with that occurring over a cavity. Hanot stated that certain solid tumors in contact with the trachea can transmit sounds very like those heard over a cavity. Bertier found this syndrome in tuberculous pulmonary sclerosis, and said that the stethoscopic signs were produced in the bronchi and transmitted intensely by the scar tissue around it. A review of 300 cases by French writers disclosed the pseudocavity syndrome in ten. Palpation of the suprasternal notch was emphasized as a means of diagnosis. In three observations published by Besançon, Jacquelin and Morin the discordance between general and functional signs on one hand and local signs on the other was pointed out. In 106 cases of tuberculosis de Martini noted tracheal deviation 38 times, with a predominance to the right, presumably because of the greater richness of connective tissue at the right apex. Other instances of pseudocavity syndrome due to syphilitic pulmonary sclerosis, serofibrinous pleurisy, and contraction of the right upper lobe from pulmonary lymphogranulomatosis are given. Tracheal deviation is part of the process of pleuropulmonary sclerosis accurately described by Laennec. The refinements of radiology scarcely alter his observation of the flattening of the chest, the retraction of the interspaces, the deviation of the verte-

bral column, and the elevation of the diaphragm that occur in these cases. All such deformities have generally been attributed to retractile pleural symphysis, but retractile sclerosis of the lung can act on the mediastinum and on the chest-wall even with a free pleura. This rôle is more apparent in destructive pulmonary lesions healed by reduction of their volume. The trachea drawn over by fibrosis transmits the normal glottic sound through the contracted pulmonary tissue to the auscultated chest-wall with amphoric or tubular quality. Three cases of nontuberculous retractile sclerosis with the pseudocavity syndrome (two of bronchial dilatation and one of pulmonary syphilis with secondary tuberculosis) are reported. Seventeen cases of tuberculous fibrosis with retraction are also detailed. Ten occurred on the left and 7 on the right, 10 in women and 7 in men. In two instances there was concomitant dilatation of the bronchi. In view of the previous statement that retractile sclerosis of the lung does not imply involvement of the pleura, the frequent coëxistence of the two processes in the present series is noteworthy. In 12 of the 17 cases total symphysis was found in 12 and partial in three. In the other three cases the state of the pleura was not determined by any attempts to administer artificial pneumothorax. It would appear that pleural symphysis, whether spontaneous or provoked by artificial pneumothorax, constitutes one of the modes of cicatrization of pulmonary tuberculosis. In 6 cases symphysis occurred spontaneously, in 6 others following artificial pneumothorax, in one following thoracoplasty and in another following spontaneous pyopneumothorax. *Conclusions:* The pseudocavity syndrome resulting from deviation of the trachea in retractile pleuropulmonary fibrosis has been demonstrated more frequently since the advent of radiology. Eighty-five per cent of the cases occur in pulmonary tuberculosis. Thoracic deformities point the way to a clinical diagnosis, especially where the general condition is good, expectoration scanty, and the temperature practically normal, but the X-ray is necessary for confirmation and exactness.—*Le syndrome pseudo-cavitaire dans les scleroses pleuro-pulmonaires, E. Sergent and N. Stoichitza, Arch. Méd.-Chir. d. l'Appar. Respir., July, 1929, iv, 297.*—(*E. H. K.*)

Haemoclastic Crisis and Tuberculin.—This study is based on observations made on 140 patients. They were divided into three groups: (1) those presenting clinical signs of tuberculosis and a positive tuberculin reaction, (2) individuals with a positive tuberculin reaction and no clinical manifestations of disease and (3) those with a negative tuberculin reaction and no evidence of tuberculosis. While fasting, the white blood cells were counted and in some cases the blood-pressure was determined. Following this 1 cc. of Old Tuberculin in dilutions up to 1:1,000,000, was injected. Every half-hour thereafter the leucocytes were again counted for two to three hours. A positive tuberculin test was obtained in 121 cases, and 85.9 per cent of this group showed a definite leucopoenia. In the majority of cases the drop in leucocytes averaged between two and three hundred. A haemoclasia was observed in 91 per cent of the patients in group 1; 88 per cent in group 2; and 11 per cent in group 3. The results obtained justify the use of this test as a diagnostic means in tuberculosis.—*Sul valore della prova tubercolino emoclasica nei tubercolotici, G. Macchiaro, Tubercolosi, March, 1929, xxi, 92.*—(*U. E. Z.*)

Rapid Diagnosis of Tuberculosis by Animal Inoculation.—Although numerous methods of cultivation of tubercle bacilli have been advanced, none has yet been devised which is as reliable as animal inoculation. Extirpation and examination of lymphoid tissue in the neighborhood of the site of inoculation leads to satisfactory results. In the last two years 150 guinea pigs have been inoculated into the popliteal lymph nodes, which are readily palpated and inoculated unless the animal be very fat. (In that event the fat may be squeezed away.) The animals were palpated twice a week. As soon as the nodes became firm and enlarged they were extirpated. This may be done quickly (in a minute or two). The hair is plucked and the overlying skin sterilized with iodine. The lymph node is held between the thumb and finger and the skin nicked; the node pops out and the incision is closed by small clamps. The pulp is smeared on slides, one of which is stained by the Ziehl-Neelsen method and the other by the Jötten-Haarmann method. Of these, the latter is distinctly better because the red tubercle bacilli are easily discernible through the picric-acid counterstain. In 30 control guinea pigs, inoculated in this way with tubercle bacilli, all yielded acid-fast bacilli. Eighty-one instances of clinical disease were used in the trial of this scheme. The earliest day of proof was the thirteenth after inoculation, but bacilli were so numerous in this instance that diagnosis might have been made earlier. By this method 11 instances of urine, 4 of pus, 2 of fistulous discharge, 2 of material obtained by joint puncture and one of lymph-node puncture yielded tubercle bacilli. In every one of these instances in

which bacilli were found in lymph nodes, the animals developed generalized tuberculosis, in no instance of negative results on extirpation was an animal found tuberculous later at autopsy.—*Beschleunigter Nachweis von Tuberkel Bazillen im Tierversuch durch Drüsenexstirpation, W. Pfannenstiel, Deutsche med. Wchnschr., December 20, 1929, iv, 2130.*—(*H. S. W.*)

Klopstock-Neuberg Antigen.—Most authors are agreed that in the serodiagnosis of tuberculosis, the deviation of the complement test is always negative and of very little diagnostic value in early cases. The exact aetiology of many sclerosing basilar lesions often is undetermined. Many children with bronchopulmonary lesions are frequently classed as tuberculous suspects because there is no way of positively determining the nature of the clinical findings. By means of the recently simplified Klopstock-Neuberg antigen, the serodiagnosis of tuberculosis has become a valuable aid in definitely determining the cause of lesions hitherto classed as doubtful.—*La serodiagnosi della tubercolosi con gli antigeni di Klopstock-Neuberg, G. F. Capuani, Tubercolosi, November, 1928, xx, 433.*—(*U. E. Z.*)

Red-Cell Sedimentation Reaction.—This test offers very little that cannot be learned by a good clinical examination. From the weight curve, temperature chart, pulse-rate, and respiratory rate, more can be ascertained concerning the patient's progress than by the sedimentation rate. Some authors state that the onset of complications can be foretold by this test. In this study it was found that the early physical manifestations were of greater value. In a tuberculous individual in good physical condition, a low sedimentation rate may indicate a good prognosis.—*Sul valore della prova di sedimentazione delle emazie nella tubercolosi polmonare, R. Doria, Tubercolosi, April, 1928, xxiii, 125.*—(*U. E. Z.*)

Primary Tuberculous Infection of Tonsil.—The general opinion is that the tonsil infected primarily with tuberculosis does not differ in gross appearance from others, and that the condition is never diagnosed until after the tonsil is seen in section under the microscope. The author presents a case in which the diagnosis of primary tuberculosis of the tonsil was made before tonsillectomy. A boy, six and one-half years of age, had had poor appetite and vague abdominal pains for two months. There had been no exposure to tuberculosis. He had always been subject to digestive disturbances, with abdominal pain and loss of appetite. One year before examination for the present illness he had had unproductive cough for four months, at which time inspiratory râles had been heard over both sides. Examination showed a pale, listless child, of average weight for height and age. Near the upper pole of the left tonsil was a single shallow ulcer, with ragged edges, and covered with dirty yellowish-gray exudate. Its area was about 1 by 0.5 cm. There was a similar, smaller ulcer on the right tonsil. Smears from these ulcers contained numerous acid-fast bacilli. Cultures were negative for diphtheria. The patient later went to a private doctor for tonsillectomy. The tonsils were not saved. One year prior to examination, his tuberculin test was negative. At the time of examination it was positive. He is now, 3 years later, clinically well. The type of tuberculous involvement of the tonsils most commonly described is that in which the germinal centres are infected, as a part of a generalized tuberculosis. In the type represented by the case here reported, the infection is confined to the lymphatic tissue immediately under the epithelium of the crypts, with concomitant ulceration by destruction of epithelium. In this type of primary tuberculous ulceration of the tonsils, the infection is obviously "open," the focus is easily removed, and probably safely so in the absence of tuberculosis elsewhere in the body.—*Primary Infection of the Tonsil with Tuberculosis, L. B. Dickey, Arch. Pediat., March, 1930, xlvii, 190.*—(*P. J. W.*)

Primary Tuberculosis of Larynx.—Although long denied by most clinicians, who were convinced of its dependence on pulmonary tuberculosis, primary tuberculosis of the larynx has been definitely recognized by all authors, but most of them regard it as rare and exceptional. Cambrelin asserts that this is not the case, that it is much more frequent than believed and often exists without being suspected, perhaps the only subjective symptom being huskiness. It is true that it is often difficult to establish that tuberculosis does not coexist elsewhere and it is better to say that there is no other manifest focus. *Characteristic Lesions:* The site of election of initial tuberculous lesions of the larynx is the posterior portion,—the interarytenoid space and the mucosa of the arytenoid cartilages. The latter is reddish violet, oedematous, sometimes ulcerated, and presents often little elevations, while the mobility of the cartilages is reduced. Besides the huskiness and rapid vocal fatigue there may be pain on deglutition. The epiglottis, however, is never involved, as it frequently is in cases associated with pulmo-

nary lesions. Furthermore, initial laryngeal lesions are generally torpid or change but slowly, while with pulmonary involvement, a source of continuous irritation, they are more apt to progress rapidly. This illustrates Louis's theory of laryngeal contamination, but this does not explain primary laryngeal tuberculosis, for which one must assume propagation by either the lymph- or blood-stream. Of course one can not absolutely deny the possibility of implantation from inhalation of contaminated dust or sputum. *Symptoms, Signs and Diagnosis:* The first indication of suspicion is usually a simple huskiness of voice, and Cambrelin now believes that both larynx and lungs of all young subjects with intractable huskiness of voice should be examined. A point of interest is that, while in the type of case under discussion the pulmonary parenchyma is not involved, roentgenographs generally show either peribronchial sclerosis or hypertrophy of hilar glands. Besredka's complement-fixation reaction has several times been positive. Cambrelin believes the natural tendency is toward the later development of pulmonary lesions if the process is not checked. Among the cases he reports of undoubted primary laryngeal involvement all have been checked by roentgenographic films, and some by pathological biopsy. Also, two other competent men examined the larynxes. *Case Reports: 1.* A young girl of 23 presented herself two years ago because of persistent huskiness and slight pain on deglutition. The larynx showed a little paresis of the left vocal cord, the interarytenoid space was oedematous, and the left arytenoid cartilage slightly hypertrophied. No bacilli were found in the sputum and pulmonary findings were negative. A few weeks later, as the hypertrophy of the left cartilage became more pronounced, a fragment of mucosa was removed and showed "subepithelial granulomatous lesions with Langhans type giant cells considered typical of tuberculosis. When this little area had healed, galvanocauterization, the only effective treatment, was begun, with three applications at weekly intervals. The patient then declared herself cured. Three weeks later she had violent pains in the head and died in a few days of tuberculous meningitis. Cambrelin has since determined never to use the deep galvanocautery in primary tuberculous laryngitis as he believes it may favor dissemination in cases where the lungs do not provide an outlet. *2:* A Pole, aged 28, complained of sharp pain in the throat and constant vocal fatigue. His interarytenoid space was velvety, the left vocal cord sluggish, and both arytenoids, especially the left, infiltrated. Pulmonary signs were negative; also X-ray films of the chest. Besredka's reaction was ++. A fragment of mucosa from over the arytenoid showed numerous mast-cell clasmatocytes and rare giant cells, diagnostic of tuberculosis. Deep galvanocauterization was performed at three sittings; the patient was then sent to a sanatorium and strict vocal rest enjoined. In six months the larynx was clear. Two years later the lungs remained intact and the case was presented before the Brussels Otolaryngological Society as cured. *3:* Three years ago Cambrelin was called to examine the larynx of a child of 4, unusually docile, whose father had tuberculosis and whose family were worried because of persistent vocal huskiness. The larynx was extremely red, the arytenoids distinctly oedematous, the mucosa of the interarytenoid space uneven and rough; in all, the larynx looked strongly suspicious. The lungs were intact, on both physical and roentgenographic examination, excepting for a little peribronchial sclerosis and tracheobronchial adenopathy. A year and a half later the child had a serofibrinous pleurisy which lasted two months and injection of a guinea pig with the fluid produced tuberculosis. After a sojourn in Switzerland and one by the sea the child's condition was much improved although it remained thin and often had an evening rise in temperature. *4:* A man of 35 had for a long time suffered from laryngitis, hoarseness and persistent dry cough. The larynx showed typical lesions,—uneven surface with swelling of the right false cord and redness of both true cords. The lungs were normal. Two months after the first observation the patient developed a right cervical adenitis, terminating in a cold abscess, puncture of which yielded a grumous fluid containing pneumococci. The mass was finally enucleated and showed caseating tuberculosis. *5:* A young woman of 33 presented herself with a rebellious cough of a year's duration, a slight aphonia, a diffuse laryngeal redness, with arytenoids swollen and the interspace velvety. The lungs were negative physically and roentgenographically. Besredka reaction was +++. She was given ultraviolet baths as she refused sanatorium treatment. *6:* A young man of 24, an amateur singer, had lost his voice for several weeks and coughed severely in the morning. His larynx at first showed evidences of acute inflammation which retrogressed after two weeks' vocal rest and palliative treatment, then showing generalized oedema and redness, involving the epiglottis. There were small mucous erosions over the arytenoids after disappearance of this oedema. He was in the hospital for a time with bronchitic symptoms but the pulmonary examination was nega-

tive. The Besredka reaction was ++. Later, during a scarlatina, the patient had violent pain on deglutition; his arytenoids were infiltrated, the right hypertrophied, the vocal cords reddened. A fragment removed from the right arytenoid showed giant-cell infiltration suggestive of tuberculosis. Three more cases are cited in which the initial laryngeal lesion appeared to evolve rather rapidly toward a pulmonary development. *7:* A woman of 37 complained of hoarseness and slight pains in the throat. She had a bitonal voice and sluggish movement of the cords, while the interarytenoid space was velvety and the arytenoids abnormally red. Her husband was a tuberculous sanatorium patient. A year later, while much better generally, she showed slight signs in the upper part of the left lung, confirmed by roentgenographs, which showed calcifications and also discrete subclavicular lesions. The other two cases were rather similar, but one developed serious cavitary lesions. *Summary:* These cases are substantiated by either biopsy, development of pulmonary tuberculosis, or characteristic lesions and Besredka complement reaction. Primary laryngeal tuberculosis, therefore, does not appear to be as rare as thought, though it may be missed. If treated early it is susceptible of cure. The sanatorium, silence, and superficial galvanocauterization are the principal measures, with perhaps the assistance of diathermy, heliotherapy and ultraviolet radiation.—*La Tuberculose primitive du Larynx, Cambrelin, Rev. Belge d. l. Tuberc., January–February, 1929, xx, 11.*—(*A. P.*)

Surgical Tuberculosis.—Conservative and operative treatment of tuberculosis cannot be sharply separated because, whenever surgery is resorted to, general measures must also be brought into play. Also, surgical tuberculosis is not to be thought of as merely local disease. It is nearly always a general disease. The relative merits of these two general types of treatment cannot be adequately evaluated except after application to large numbers of cases, in which very marked difference in the two groups of cases must be clearly obvious. Merely to shorten the time (duration) of treatment should not of itself be a leading indication as to whether conservative or surgical treatment should be instituted. Judgment of the degree of improvement is difficult because of the tendency to relapse. There is also a great individual variation in people, which must likewise be taken into account. It is not correct to compare surgical with sanatorium treatment because the latter may be more general and may include the former.—*Ueber chirurgische Tuberkulose, H. Kuss, Deutsche med. Wchnschr., March 7, 1930, lvi, 395.*—(*H. S. W.*)

Treatment of Surgical Tuberculosis by Finikoff's Method.—Intramuscular injections of one cc. of an iodide incorporated in 10 cc. of sterilized peanut oil are given every seven days. These provoke a strong leucocytosis. The mononuclear cells by their lipolitic powers destroy the waxy capsule of the tubercle bacillus. The polymorphonuclear leucocytes, because of their proteolytic powers, destroy albuminous substances contained within the protoplasm of the bacilli. Destruction of the capsule liberates fatty acids, which are deleterious to the body and cause a decalcification of the organism. To combat the noxious acids, calcium salts (calcium chloride) are given. The patients receiving this form of treatment manifest no ill effects and, in children, the results have been most satisfactory. Many patients suffering from tuberculous adenitis, tuberculous ulcerations and osteoarthritis have shown remarkable improvement.—*Résultats obtenus par la méthode de Finikoff dans le traitement des tuberculoses chirurgicales, Amnès, Prog. Méd., January 12, 1929, no. 2, 59.*—(*U. E. Z.*)

Physiotherapy of Genitourinary Tuberculosis.—A nephrectomy for tuberculous disease of the kidney was done in a patient with an advanced pulmonary lesion. He later developed tuberculous orchitis and stricture of the ureter. High frequency, electrolysis, and ultraviolet-ray treatments were followed by marked general improvement and definite betterment in the local lesions.—*Lésions tuberculoses de l'urèthre, de la vessie et des uretères. Traitement physiothérapique, Schmitt, meeting Soc. de médicine de Paris, December 29, 1928, reported in Prog. Méd., January 12, 1929, no. 2, 81.*—(*U. E. Z.*)

Pathological Anatomy of Addison's Disease.—In the original description of the disease which bears his name, Thomas Addison defined it as "a remarkable progressive feebleness of the patient without any apparent known cause, or as it is styled, asthenia, a discoloration of the skin and a disease of the suprarenal capsules." Wilks (1862 and 1865), who studied the pathology of Addison's first cases and reported others to a total of 33, described the suprarenal glands in the series as scrofulous and totally destroyed. Because the necrosis in the glands did not resemble the necrosis ascribed to tuberculosis at that time, he was rather of the opinion that the glands presented a unique disease not identical with other tuberculous

processes. Wilks's excellent description of the gross aspect of the suprarenal glands, however, leaves little doubt that the lesions were the same as those that have been seen many times since then and identified as tuberculosis by histological study. In 1892 Lewin made an extremely comprehensive review of the literature up to that time. In 1907 Hedinger reported a series of 15 cases of Addison's disease, in 14 of which there was tuberculosis and in one case atrophy of the suprarenal glands. The largest recent series was reported by Coneybeare and Millis, and comprised 29 cases with necropsies, observed at Guy's Hospital between 1904 and 1923. In the past 18 years at the Mayo Clinic necropsies have been performed in 28 cases in which the clinical syndrome of Addison's disease has been present. A review of the protocols and a reëxamination of all the material available from these 28 necropsies form the basis for the present study. In 25 of the 28 cases there was a lesion of the suprarenal glands which on histological examination was called tuberculosis. In the sections of all of these there were typical areas consisting of tubercles with endothelial cells, giant cells, fibroblasts and lymphocytes. Acid-fast bacilli morphologically resembling tubercle bacilli were found in these sections in 11 cases. The tuberculosis was found to be bilateral in all the 25 cases, and involved the entire gland, with almost complete destruction. The type of lesion varied between two extremes, a very proliferative type with many tubercles, many fibroblasts and connective-tissue cells and only small areas of necrosis, and a type in which the gland was a mass of necrosis surrounded by a fibrous capsule in which there were only a few tubercles and a small number of lymphocytes. The lesions were essentially proliferative with many tubercles and little necrosis in 7 cases; proliferative and necrotic with a moderate number of tubercles and a moderate necrosis in 8 cases; essentially necrotic with extensive encapsulated necrosis with few tubercles in 8 more cases; and inactive accompanied by encapsulated necrosis with or without calcium deposits and a few foci of lymphocytes in 2 cases. Grossly the necrosis differed from the ordinary caseous necrosis of tuberculosis in that the necrotic process was yellow or yellowish gray, firm and rubbery, and on section presented a uniform surface. As a rule the glands were enlarged and were not smaller than normal in any case. From the appearance of early and late tuberculous lesions of the suprarenal glands one type of lesion apparently encounters active resistance and much cellular proliferation which, together with some necrosis, gradually destroys the glands. Another type encounters little resistance; areas of necrosis gradually spread until the gland is practically destroyed and the necrotic mass becomes encapsulated. There are intermediate types, but the tuberculous process is always progressive until the gland is almost completely destroyed. The remaining 3 of the 28 cases of Addison's disease had advanced bilateral atrophy of the suprarenal glands, resulting from some toxic agent or some other unknown cause. Healed tuberculous lesions of the lungs were found in all the cases in which there was tuberculosis of the suprarenal glands. Active tuberculosis of other organs was found in 22 of these. In 10 there was active pulmonary (without genitourinary) tuberculosis, in 4 there was active genitourinary (without pulmonary) tuberculosis, in 2 there was both active pulmonary and genitourinary tuberculosis, and in one case there was active tuberculosis of the spine without pulmonary or genitourinary disease, all of which presumably antedated the suprarenal disease. Of the remaining 8 cases, in 3 active tuberculosis of other organs was not found and in the other 5 there was active tuberculosis of the liver, spleen or abdominal lymph nodes, all of which was considered as coincidental or secondary to the suprarenal disease. There was no active tuberculosis in the 3 cases of suprarenal atrophy. The weight of the heart was less than the average normal in 14 of 20 cases in which data were available. The remaining 6 cases showed a weight greater than the average normal. Diffuse degenerative changes in the renal tubules were present in 9 cases. In 10 cases, including the three of suprarenal atrophy, the thymus was enlarged and there was hyperplasia of lymphoid tissue in the nodes and intestinal tract. There was scattered lymphoid hyperplasia in 8 other cases. Because of the reports in the literature of cases of Addison's disease in which the suprarenal glands presented other lesions than tuberculosis and atrophy, a study was made of all the suprarenal lesions found at necropsy at the Mayo Clinic during the last eighteen years. None of the patients in these cases had clinical Addison's disease. This group of 73 cases included the following: primary suprarenal neoplasms, 15; secondary suprarenal neoplasms, 39; atrophy, 2; amyloidosis, 3; infarction, 2; haemorrhage, 3; abscess, 1; and tuberculosis, 8. In these cases the minimal approximate amount of suprarenal tissue remaining was 10 per cent. This is noteworthy since in only one of the cases of tuberculosis with Addison's syndrome as much as 10 per cent of suprarenal tissue remained; in all the other cases there was considerably less than this. If it is considered that suprarenal insufficiency is

the cause of Addison's disease it would appear that the limit of the margin of safety is reached when somewhere between 80 and 90 per cent of the suprarenal tissue has been destroyed. Possibly the reasons why neoplastic tumors so rarely produce Addison's disease are that it is unusual for the patient to live long enough to have tumors in both glands, and, if he does, the primary disease or metastasis to other vital organs causes death before the suprarenal tumors have grown large enough to destroy much suprarenal tissue. Although malignant tumors are invasive, they are not nearly so destructive to highly specialized glandular tissue as is tuberculosis.—*The Pathological Anatomy in Twenty-Eight Cases of Addison's Disease, N. W. Barker, Arch. Path., September, 1929, viii, 432.*—(*J. S. W.*)

Diagnostic Difficulties in Tuberculous Meningitis.—A seven-year-old child, convalescent from bilateral otitis media complicating scarlet fever, developed complete anorexia and pain in the neck. This was thought to be an exhaustion phenomenon from the recent prolonged illness. Next day, severe pain appeared in the head, and there was stiffness of the neck and great exhaustion. Later, speech became affected and ataxia, profound drowsiness and loss of abdominal reflexes developed, but there was no fever and no loss of consciousness. Lumbar puncture yielded a turbid fluid, with many lymphocytes but no tubercle bacilli. At the fourth puncture no fluid was obtained. Cisterna puncture yielded similar fluid, with many lymphocytes but still no tubercle bacilli, even after the most careful search. On the eighth day tube-feeding was resorted to, and the pulse was irregular. After this day there was a definite retrogression of all symptoms. Three weeks after the onset, the child walked, but did so with a left-sided spastic ataxia and had a violent attack of vomiting, followed by a brief return of all symptoms. Two months after the onset the child had returned to an entirely normal state. The differential diagnosis had to be made between abscess of the brain, tuberculous meningitis, and postscarlatinal encephalitis. The tuberculin reaction was absolutely negative in the early days of the illness; the fluid was such as might be found in an encephalitis; these facts and the absence of tubercle bacilli all seemed to point rather to a postscarlatinal encephalitis with marked symptoms of meningismus. The clinician should always bear in mind that meningismus from several causes may simulate tuberculous meningitis. Such must have been the true condition of affairs in many cases of so called "healed" tuberculous meningitis.—*Differentialdiagnose der Meningitis tuberculose, L. Langstein, Deutsche med. Wchnschr., December 20, 1929, lv, 2138.*—(*H. S. W.*)

Miliary Tuberculosis with Cerebral Arterial Thrombosis.—A female child, three years of age, previously apparently healthy, with no family history of tuberculosis (the father and brother were subsequently shown to have the disease), suddenly had a convulsion, with physical signs pointing to lobar pneumonia with meningismus. The lumbar puncture on admission produced a spinal fluid negative except for markedly increased pressure and the presence of 5 lymphocytes per cu. mm. At the end of three weeks, the patient had intermittent elevation of temperature, partial paralysis, loss of vision, hyperactive reflexes, persistent rigidity, positive Kernig. At the beginning of the fourth week, the spinal fluid was found to contain 540 leucocytes, of which 84 per cent were polymorphonuclear. Pressure was markedly increased. By the fifth week there was marked papilloedema. There were 25 lymphocytes per cu. mm. of spinal fluid. No tubercle bacilli were found. The child died during the fifth week. A diagnosis of brain abscess in the temporoöccipital region was made. The abscess was considered to have followed an encephalomalacia resulting from a septic embolus, causing a thrombosis of the right posterior cerebral artery and secondary to an initial bacteraemia or purulent otitis media. It was only late in the course of the disease that a presumptive diagnosis of tuberculous meningitis was made. Necropsy demonstrated the presence of a miliary tuberculosis with complicating tuberculous meningitis, tuberculoma of the left occipital lobe, and a septic thrombosis of the right posterior cerebral artery with resultant encephalomalacia in the right occipital lobe.—*Miliary Tuberculosis Complicated by Thrombosis of the Posterior Cerebral Artery and Tuberculous Meningitis in a Child Three Years of Age, J. S. Leopold and J. L. Rothstein, Arch. Pediat., February, 1930, xlvii, 77.*—(*P. J. W.*)

Newer Treatment of Tuberculosis.—Perhaps more than 200,000 infants have received protective inoculation with *BCG*. Sufficiently exhaustive investigation of the individual case has, however, apparently been neglected, and routine postmortem examination has not excluded tuberculosis. In certain instances guinea pigs inoculated with *BCG* have died of tuberculosis. The question of allergy in connection with artificial immunization is yet undecided. Intimate contact with the open case is the most

constant aetiological factor in phthisiogenesis. In Chicago all contacts, adults and children, are supervised. In 1927 and 1928, 9,891 new contacts were examined, and 645 cases of tuberculosis found. Of these, 545 were cases of pulmonary tuberculosis, 93 were lymphatic tuberculosis, and 7 were osseous and other forms of tuberculosis. At the end of 1928 there were 3,167 positive sputum cases under surveillance. Of these only 25 were in contact with children, and they were in the process of clearance. During the year there were 942 cases in contact. Contact was broken in 917 cases and 1,927 children thus protected from further infection. It is estimated that there are in this country about 40,000 cases of advanced tuberculosis who could not receive benefit from routine treatment or artificial pneumothorax and whose prognosis is very grave. Thoracoplasty offers much for this type of case. Crushing or removal of the phrenic nerve may greatly benefit the early case, and, according to the author's experience, will supplant pneumothorax in about 80 per cent of cases in which previously pneumothorax had been indicated. The success of thoracic surgery depends upon the surgeon's skill, the proper selection of cases, and the ability of the sanatorium to give treatment to surgical cases equivalent to that given in general hospitals. The latter provision also allows for the proper treatment of any surgical complication of tuberculosis. New work done both in Germany and in the United States indicates that the proper ratio of minerals and vitamines in the diet is essential for the healing of tuberculosis. Rest, fresh air, and good food still form the heart of the problem of therapy, and are only supplemented by the newer methods of treatment.—*Newer Methods in Tuberculosis Therapy, B. Goldberg, Ann. Int. Med., March, 1930, iii, 910.*—(*R. M. F.*)

Exercises in Pulmonary Diseases.—Massage of the limbs prepares the body for active exercise by circulatory stimulation. Movements of flexion, extension and rotation of the spine, and all respiratory exercises help secure a mobile thorax, a prerequisite to treatment of the thoracic contents with exercises, such as follow: *1:* Quick strokes on the chest-wall aid in loosening tenacious mucus. *2:* Expiration is aided by "chest shaking" (alternate pushing and pulling against the thorax). *3:* Controlled and localized breathing, by stressing the diseased lung, encourage it to expand. *4:* Alteration of posture aids in drainage of cavities. Temperature and pulse-rate are used to guide treatments in suspected tuberculosis and in pulmonary disease without an open wound. When the myocardium is not diseased a pulse-rate increase of from 20 to 40 beats per minute which returns to normal within 3 minutes after exercise is safe, and such exercise attains desired results by increase of the respiration. Cases suitable for physical therapy are divided into groups, as follows: *1:* Those in which expiration needs aid (emphysema, asthma, bronchiectasis). *2:* Those in which inspiration needs aid (empyema, pulmonary collapse). *3:* Those in which both expiration and inspiration need aid (chronic bronchitis, pneumonia with delayed resolution). In emphysema and asthma exercises should be aimed at correction of posture, the object being to reduce the bulk of the lungs and increase the power of the expiratory muscles. The same treatment is used for bronchiectasis, and postural drainage and expiratory "chest shaking" are added. In empyema exercises are begun twenty-four hours after operation, in order to promote lung expansion with adequate drainage, and to prevent scoliosis. Absorption of toxins is decreased and secondary operations become rarer. Pyrexia does not contraindicate exercises provided drainage is free. Breathing exercises and postural correction, limb massage, active arm, leg, and trunk movements are instituted, gradually, in the order named. Manual manipulation of the chest is avoided while there is an open wound. The same scheme of exercises is used in treating delayed resolution. The object is to prevent bronchiectasis and pulmonary fibrosis by stimulating the pulmonary circulation through increase in respiration. In influenzal pneumonia the patient should have been afebrile one week before exercises are begun. The persistent low-grade pyrexia of bronchopneumonia may be abolished by exercises. Results are good if physical therapy is instituted soon after termination of the acute illness.—*A Note on Remedial Exercises in the Treatment of Pulmonary Diseases, A. H. Douthwaite and H. S. Angove, Lancet, February 15, 1930, ccxviii, 346.*—(*R. M. F.*)

After-Care of Tuberculosis Patients.—The after-care of the consumptive presents an entirely different problem from 15 years ago. Formerly the healing of large pulmonary cavities with ordinary rest was rather an exceptional achievement. Now, due to improved surgical technique, a better selection of patients and a wider use of the various mechanical forms of collapse therapy, a far greater number of patients are returned to normal life. At the Laennec Hospital there is a well-organized follow-up routine between the hospital and the dispensary, and the patient's family and his employer. After a

period of hospital care, the pneumothorax patient is strongly advised to continue his refills at the dispensary. Should any complication arise he is at once readmitted to the hospital. Thus a continuity of medical control is maintained. The criteria for the return of a pneumothorax patient to his occupation are freedom from symptoms for about a year and a negative sputum which does not infect a guinea pig. Farming, contrary to popular belief, is too strenuous an occupation for the arrested tuberculosis patient. Every effort should be made to have him return to his former occupation and to give him light tasks until his pulmonary lesion shows stability or further retrogressive changes.—*The After-Care of the Consumptive Patient, E. Rist. Brit. J. Tuberc., January, 1930, xxiv, 7.*—(*J. G.*)

Technique of Thoracoplasty.—The treatment of vast pulmonary cavities is not the sole indication for extensive thoracoplasty. Certain pleural complications of artificial pneumothorax, especially pulmonary perforations, occurring either in an uninfected pleura or in one already containing purulent exudate, with or without secondary infection, may need this intervention. The same thing is true of bronchial perforation into an oleothorax, a complication which should be well known and dreaded by those using oleothorax. Although such a perforation may undergo spontaneous cure, usually when complicated by pleural suppuration and infection it leads to a tuberculous generalization, with hectic fever, and demands surgical intervention which must come early if it is to be of avail. When one is dealing with simple purulent or puriform exudates, following the subsidence of ordinary tuberculous pleurisies, well described by Bernard and Baron under the term "exudative pleurisies of artificial pneumothorax," thoracoplasty does not seem necessarily indicated, even though it may be curative. As good cures have been obtained in inveterate cases without resort to it. For example, Renaud recommends in such cases pleurotomy with drainage, while Bernard, Kindberg, Rist and Burnand are decisively opposed to this. All these report good and lasting results from repeated puncture, followed by injection of air, and more recently by oleothorax. In cases with secondary infection the situation is more serious. There are doubtless forms, especially staphylococcic, of low virulence, in which the general condition is not gravely affected, there is only moderate fever, and cure may result from simple measures; but these are rare. Secondary infection of pneumococcic type may also result from a bronchopulmonary infection and these may be expected to terminate badly. Ordinarily a parietal fistula or perforation of a cavity calls for prompt intervention and cannula drainage is merely a temporizing measure, although it sometimes results in temporary amelioration. Anterior drainage is particularly unsatisfactory; even with rib-resection it is insufficient because poorly located, and without it compression by the costal cartilages and thickened pleura will usually cause blockage within a few hours. Neither will anterior pleurotomy prevent involvement of the skin near the thoracoplasty incision since if the operation is extensive an anterior incision also will be required. The following method, after trial of many, is recommended: In case of pleural suppuration one first has recourse to repeated punctures, continuing them through successive stages of thoracic resection and, in certain cases, a pleurotomy practised only after cicatrization of the posterior thoracoplasty, since the incision should be at the lowest point postero-inferiorly. Surgical treatment should answer two indications: *1:* It should suppress as completely as possible the suppurating pleural cavity, by extensive bony resection and falling together of the walls of the cavity. *2:* It should maintain the collapse of the tuberculous lung, reëxpansion of which might awaken fresh activity. Thoracoplasty is relatively easy in recent cases of suppuration, but very difficult in cases with long-standing changes in the pleura and chest-wall, including narrowing of the intercostal spaces and adhesion of the ribs, especially the upper ones. In some cases there is a pachypleuritis of almost cartilaginous consistency, adherent to the periosteum, making denudation of the ribs very difficult. In such cases the operation must be divided into more stages and supplemented by an anterior one. Another difficulty is parietal fistulization following repeated punctures of a suppurating pleural cavity. These fistulae or infected portions of the chest-wall should be touched only after closure of the main wound, when they may be opened and drained. Küss has emphasized the danger of letting a lung stay bathed indefinitely with purulent richly bacilliferous fluid, which is likely to lead to tuberculization and progressive cortical ulceration, and finally to perforation. Perforation may be diagnosed by the functional and general symptoms, the sudden acute dyspnoea, thermic rise, recrudescence or appearance of pleural exudate, and confirmed by the manometric findings or expectoration of liquid, and a very active therapeusis is indicated. It is understood that we speak only of large permanent perforations, the symptoms of which become aggravated rather than ameliorated, and not

of the small transitory perforations and fissures which Bard believes so frequent, that is, that all pneumothorax exudates indicate such occurrence, which opinion, though doubtless extreme, contains much truth; latent perforations are certainly more common than Bard's contradictors believe. Sometimes a perforation is complicated within a few days by a miliary dissemination in the other lung, doubtless due to bronchial embolism. Doubtless such dissemination is favored by the incessant violent coughing, which accompanies a partial bronchopleural fistula, and repeated pleural punctures should then be carried out. Before fistulization is established punctures are only necessary to avoid overpressure of the liquid in the pleural cavity, which the bony resections have reduced and which otherwise may lead to asphyxia by bronchial inundation. When frequent punctures become necessary to prevent the spitting of pus, the time has arrived for a complementary pleurotomy. It is recognized that such a wound will be long in healing and subject to fistulization, but between two evils one must choose the lesser. Posterior pleurotomy following thoracoplasty is also not easy technically. The incision goes through a lardaceous tissue, and a thickened cicatrix undergoing secondary ossification, where it is practically impossible to avoid cutting intercostal vessels. A needle should first be thrust into the pus-cavity and will traverse a veritable carapace of 2 to 3 cm. One may select the terminal part of the posterior thoracoplasty incision, near the eleventh rib. An elliptical incision is made around the needle, leaving it in place as a landmark. A minimal incision is made with a bistoury and forceps, and a drain of about 1 cm. diameter inserted and connected by glass tubing to another drain leading to a bottle near the bed. In certain cases a Bulow's cannula may be substituted for the needle and the tubing connected with this. In cases like these total thoracoplasty must be done while they are "hot" if at all. One intervenes on the diagnosis of a serious complication, such as infected exudate or perforation. Temporizing diminishes the chances of success. It is the duty of both physician and surgeon to accept the inevitable risks. Only an acute contralateral extension should constitute a serious contraindication. Although not all those operated on under these conditions can be saved, the failures should not be attributed to the intervention, which has proved its value. (Note: Rist adds that certain cases of infection must first be drained. Rolland and Maurer think, with Archibald, that in such cases thoracoplasty has a poor chance. Bordet also states that in certain cases pleural punctures are so difficult as to be impracticable and pleurotomy must be done. In these cases one's hand is forced).—*Indications de la thoracoplastie totale élargie dans le traitement des pneumothorax compliqués (suppurations pleurales et perforations pulmonaires), J. Rolland and A. Maurer, Section d'études scientifiques de l'Oeuvre de la Tuberculose, January 12, 1929, reported in Rev. d. l. Tuberc., April, 1929, x, 247—(A. P.)*

Technique of Thoracoplasty.—It was Boiffin who first showed that posterior costal resections of equal size led to more important reduction in size of the thoracic cavity than anterior or lateral ones. This has since been thoroughly confirmed. Maurer here reports the results of 25 thoracoplasties (38 operations) done by him over a period of three years. *Technical Rule No. 1:* The rib must be resected at the point of contact of the costotransverse articulation. *Rule No. 2:* Local and regional anaesthesia should be used. Maurer has never had to resort to a general anaesthetic nor had a patient refuse a second operation under local anaesthesia. There has been only one case of superficial suppuration of the chest-wall; in every other case primary reunion of the tissues has occurred and the stitches were removed after a week as in a laparotomy. No phenomena of intoxication have been noted, using a 1–200 novocaine-adrenalin solution (Reclus's solution) and not employing over 80 cc. (less than 0.5 gm. novocaine) for a resection of 6 ribs. To obtain the best results, the surgeon should be familiar with the clinical forms of tuberculosis, that he may discuss with the medical specialist surgical methods, the best time for intervention, its division into stages, and whether thoracoplasty should be partial or complete, and begun with the upper or lower ribs. Maurer's cases were all carefully examined by Rist or his associates. Rolland, who is particularly familiar with surgical methods, assisted at all operations and watched the patients during the ensuing days. To this careful cooperation is attributed the very low mortality, a single death out of 38 operations on 25 patients. This case succumbed to a dissemination of the disease in the lower parts of his lung and a generalization which occurred about three weeks after operation. This case had previously been in active evolution and it now seems that instead of a superior thoracoplasty a total one in two stages, beginning from below, might have resulted more favorably. *Extent of Costal Resections:* These should increase progressively from above down, from the first to the tenth rib. About 2 to 3 cm. of the first rib

are removed, 6 to 8 cm. of the second, 12 to 16 cm. of the others, and 8 to 10 cm. of the eleventh rib. However, the extent of resection is a function of the clinical forms and of the subject's resistance. Generally, in the absence of pleural exudates, resections should be more extensive,—18 to 20 cm. of the lower ribs. Posterior resections may in some of these be supplemented by anterior resections. Total thoracoplasty ordinarily requires two stages or more, allowing not over two weeks to elapse between the different stages. *Technique:* The position of the subject is very important, in order that both he and the operator may be at ease during a long delicate procedure and no fatigue or impatience result. The subject is placed on the table in lateral decubitus resting on the sound side, with a cushion under the lower ribs and costoiliac space. A second cushion is placed in front of the lower thorax and belly in such a way that the part nearest the head receives the elbow. The hand on the same side (the diseased) rests on a roll which supports the lateral concavity of the neck, while the head rests on a thinner cushion. One may fold the other arm under the head and place it between two cushions or rolls. All these things are illustrated by drawings. Sometimes, when the patients have voluminous secreting cavities or pulmonary perforation with infected pleural exudate, lateral decubitus on the sound side provokes spasms of coughing which may spread the process in the other lung. In such cases a more or less sitting position is preferred, arms folded and crossed in front of the chest, the head supported. Disinfection of the skin is carried out with 5 per cent iodine over the hemithorax, shoulder and axillary folds. The vertebral column and iliac crests are marked out. The supraclavicular space is also disinfected up to the roots of the hair. Local and regional anaesthesia is employed altogether, local for skin and muscles, regional for the intercostal nerves. For intracutaneous anaesthesia a short-bevelled needle about 4 cm. long and a 2 cc. hypodermic syringe are used. A series of intracutaneous "buttons" is made. For the muscular masses one uses a 10-cc. syringe, injecting with a to-and-fro movement, and then progressively withdrawing the needle while continuing to inject. Cutaneomuscular anaesthesia completed, the skin and cellular tissue are sectioned, exposing the plane of the trapezius, whose fibres run horizontally in the upper portion and obliquely upward and outward in the part attached to the inner scapular margin. A slightly curved incision will best expose the underlying structures, but a nearly vertical incision, less extensive upward, yields far better aesthetic results and shoulder function. The trapezius having been sectioned, a second plane, consisting of rhomboids and levator angulae scapuli, is exposed. The fibres of the rhomboids are directed downward and outward toward the internal scapular border, below its spine. This plane is cut about 2 cm. from the internal scapular border. The posterior scapular vessels are situated in a fibro-fatty layer and are sectioned between ligatures with catgut. Small muscular arteries should be clamped and tied at once. This helps to avoid operative shock. Some of the sacrolumbar or iliocostal muscle masses must be retracted in order to expose the inner side of the ribs and costotransverse articulations. To do this without pain, several cc. of cocaine are injected near the external border of the common mass prior to separating the small tendinous aponeuroses from the bone. Regional anaesthesia of the intercostal nerves is practised by means of a needle 5 to 6 cm. long attached to a 5-cc. syringe. If one intends to begin by resecting the fifth rib, for instance, the sixth, fifth and fourth nerves are first anaesthetized, then the third, and so on up to the first. The needle is introduced obliquely at 45° in contact with the lower border of the costotransverse articulation, directing the point downward and inward and is then thrust to a depth of 2 cm. into this space between the ribs. There may be slight pain. About 4 cc. of solution are injected about the nerve, which may distend somewhat the posterior part of this space. The higher the anaesthesia is carried, the nearer the lower border of the rib one must go. Incision of the periosteum and denudation of the ribs are described; the latter begins inferiorly over an extent of 8 to 10 cm. Then come section and extirpation. Denudation of the internal aspect of the first rib must be done under finger control. One must avoid going too far out because of the proximity of the posterior scapular artery and cords of the brachial plexus. If this artery is cut it is very difficult to clamp and one may have to resort to tampons. Fatal haemorrhage has resulted. Finally haemostasis is checked up and made perfect and a long fenestrated drain of the thickness of a finger is inserted. When this is put in place the various layers are sutured and finally the skin. A few flat gauze compresses are the only dressing. *Postoperative Care:* The patient is put to bed in a sitting position with pillows behind and on each side. An injection of pantopon and camphorated oil is given at once and digitalin started. The camphor in oil is repeated four or five times in twenty-four hours, also the pantopon each night. The stitches are removed on the eighth to tenth day. *Incidents and Accidents:* Among

these are haemorrhage from the posterior scapular artery, superficial suppuration, and gas embolism. Maurer reports one case of the last in which he saw the open lumen of the vessel (intrathoracic) and heard a slight sucking sound. The patient went into syncope, he applied a compress to the vessel and recovery ensued with only a sense of malaise. No deaths from operative shock occurred but one did occur the third week from dissemination in the lower lobe from an actively secreting lesion.—*Technique de la thoracoplastie extrapleurale paravertebrale pratiquée sous anesthésies locale et régionale dans le tuberculose pulmonaire, A. Maurer, Section d'études scientifiques de l'Oeuvre de la Tuberculose, November 10, 1928, reported in Rev. d. l. Tuberc., February, 1929, x, 96.*—(*A. P.*)

Technique of Thoracoplasty.—In these cases one must not be content with too parsimonious bony resections, as they are absolutely insufficient, the pulmonary obliteration is minimal, and the cavities persist only slightly diminished in size. Bernou and Fruchaud have published their observations and films showing the need of more extensive resections which they now practise. This branch of surgery has undergone a definite evolution. It was first necessary to show that the patients operated on could survive and then to put them in as favorable conditions as possible for ultimate cure. Total thoracoplasty, practised according to the technique laid down and including all the ribs posteriorly outside their costotransverse articulation, will usually bring about a sufficient reduction in chest volume. This reduction, which continues following operation, is usually large enough if one takes care to remove (in a man of ordinary height) from 1.3 to 1.45 metres of ribs, but with a voluminous cavity in the apex or middle part of the lung, even more extensive costal resections are required. In such a case third and sometimes fourth operations may be necessary in order to obliterate the anterior arches of the ribs already resected posteriorly, and little by little one approaches a deossification of the chest-wall. These voluminous cavities may occur in lungs previously treated by artificial pneumothorax too soon obliterated by adhesions. The appearance or increase of a pulmonary cavity in a symphysized lung is presumptive proof of reactivation of the lesions and an indication for intervention. In other cases a thoracoplasty should be contemplated in the beginning, with no preliminary pneumothorax. When a cavity has destroyed the lung over a large extent in contact with the chest-wall, when it is limited by a thin shell which lines the apical pleural dome, one should not attempt pneumothorax. Even if a pulmonary separation is realized it can be no more than partial and usually ceases where the cavity begins. Besides, these excavated forms of tuberculosis treated by pneumothorax are particularly exposed to the dangers of pulmonary perforation. Oleothorax under pressure is even more likely to hasten a fatal perforation. At the same time, these giant cavities are bad cases for thoracoplasty. The ribs are intimately bound to a very rigid fibrous layer, constituting the cavity wall. In places this fibrous tissue is sown with soft fungosities, indicating parietal infiltration by the tuberculous process. The rigidity of the deossified wall may give rise to dead spaces where serosanguineous fluid accumulates and is easily secondarily infected. Roentgenographic pictures of these voluminous cavities with rigid walls often show at their lower pole a horizontal level indicating stagnant residual fluid which can only be evacuated through the bronchus of drainage. This stagnant fluid is favorable to secondary infection. Septic retention manifests itself by a thermic ascension and may set up either a localized bronchopneumonia or a generalized septicaemia. In some cases it is reasonable to look for complete closure of such a voluminous cavity. The different stages of operation should not be too far apart. The optimum interval is about two weeks. Likewise when an anterior complementary operation is decided on to supplement a posterior thoracoplasty it should be carried out within two weeks of the last stage of the latter. *Technique:* Under local anaesthesia one traces a line by a series of intracutaneous buttons juxtaposed along the length of the inferoexternal border of the large pectoral muscle, ending above and outside the anterior surface of the tendon; in order to avoid producing a cicatricial band in the axilla. The edge of the exposed pectoral is retracted upward and inward, its relaxation being secured by injecting about 5 cc. of novocaine about 1 cm. below the lower border of the middle of the clavicle. The nerve to the large pectoral is anaesthetized where it crosses the axillary artery. The small pectoral is then exposed and the nerve to the serratus magnus. Beneath the cellular tissue are the muscular digitations. The intercostal nerves are anaesthetized by directing obliquely forward from behind, guided by the finger, a needle mounted on a 5-cc. syringe filled with 1–200 novocaine. The lowest ribs are resected first. The anterior cartilaginous junctions are cut with a bistoury, while posteriorly they are enucleated after removal of the periosteum. The second, and especially the first, ribs are difficult to extirpate and one may have to cut trans-

versely the tendon of the small pectoral muscle. A resection of 2 cm. of the first rib is sufficient, as 3 cm. have already been removed posteriorly. The superior aspect is first stripped of periosteum, then the inferior, and this is verified by palpation with the left index finger. The pectoral muscle is reunited, a drain placed in the axilla and the inferoexternal border of the great pectoral fixed outside the cellular tissue. The drain is ordinarily removed in three days. Excellent drawings accompany this paper.—*Indications et technique de la thoracoplastie élargie dans le traitement des cavernes volumineuses du poumon, J. Rolland and A. Maurer, Section d'études scientifiques de l' Oeuvre de la Tuberculose, December 8, 1928, reported in Rev. d. l. Tuberc., February, 1929, x, 129.*—(*A. P.*)

Anaesthesia for Thoracoplasty.—From experience with 101 thoracoplasties, representing 202 operations, at the Brompton Hospital, it is concluded that the best results are obtained with nitrous oxide and oxygen anaesthesia, combined with block of the intercostal nerves with 1 per cent novocaine as soon as the ribs are exposed. Ethylene has not been found to be vastly superior, though there is an advantage in the fact that anaesthesia can be maintained with a greater percentage of oxygen than is possible with nitrous oxide. In certain cases a general anaesthetic may be definitely contraindicated, but as a rule it is best to use it if possible, because there is less liability to shock. With nitrous oxide and oxygen it is always necessary to give efficient preliminary medication. A combination of omnopon, gr. 1/3, and scopolamine, gr. 1/150, has been found useful, and does not make it difficult for the patient to clear his lungs of sputum.—*Anaesthesia for Thoracoplasty in Pulmonary Tuberculosis, I. W. Magill, Lancet, February, 8, 1930, ccxviii, 295.*—(*E. H. K.*)

Immediate Risks of Thoracoplasty.—Dumarest emphasizes especially the difference in the operative risk according to whether the thoracic cavity is empty or contains lung. Pleural indications for operation are quite frequent: septic pleural suppurations, with or without pulmonary fistulization, when attempts at disinfection by lavage have failed or the fistula persists. The only hope then is obliteration of the pleural cavity, executed in several stages, preceded by external drainage. Such intervention may be undertaken with less apprehension than in the case of a thoracoplasty for pulmonary indications. The most grave immediate risks of bony resection are due to: *1: Mechanical Causes:* Displacement or fluttering of the mediastinum, mechanical embarrassment of the heart, whence syncope or asphyxia may ensue. *2: Infectious Causes:* Inoculation of the healthy lung by bronchial emboli carrying septic products, whose elimination by expectoration is difficult. When the pleura is empty, neither mediastinum nor heart, nor respiratory dynamics, are influenced and the effects are parietal rather than visceral. There remains operative shock: it may occur in solely pleural interventions, but more rarely. A pleural thoracoplasty is also easier to perform and its indications are broader than when done for pulmonary disease.—*Sur les risques immédiats de la thoracoplastie, F. Dumarest, Section d'études scientifiques de l'Oeuvre de la Tuberculose, January 12, 1929, reported in Rev. d. l. Tuberc., April, 1929, x, 255.*—(*A. P.*)

New Thoracoscope.—One instrument serves the double purpose of endoscope and active electrode. Its advantages are as follows: *1:* Its small calibre (18 Charrière gauge). *2:* Its working through a cannula. *3:* The need for only one puncture. *4:* The excellent field of vision, which is at an angle of only 30° off the axis, thus simplifying orientation. *5:* The advantageous position of the electrode just beyond the lamp, making it as easy to touch as to see an adhesion. *6:* The fact that it is a diathermy instrument. A suitable adhesion favorably located, can be first coagulated and then cut through with no production of smoke. *7:* Easy sterilization of the whole instrument by boiling. The principal innovation is making the telescope itself carry the coagulating and cutting current. A detailed technical description of the apparatus is included in the article.—*A New Thoracoscope, F. G. Chandler, Lancet, February 1, 1930, ccxviii, 232.*—(*E. H. K.*)

Sudden Death Following Phrenicectomy in Diabetic.—The operation was performed for bronchiectasis. It is sometimes advisable to operate with the patient sitting up in order to avoid infecting the healthy lung. Rist states that in spite of the reported deaths, phrenicectomy is a valuable therapeutic agent and has given excellent results in many cases. A few fatalities should not be the cause of discarding the operation. *Sur un cas de mort subite après phrenicectomie chez un diabétique, Burman, Sergent and Rist, meeting Soc. Méd. des Hôp., January 18, 1929, reported in Prog. Méd., February 2, 1929, no. 5, 199.*—(*U. E. Z.*)

Bilateral Phrenicotomy.—Bilateral phrenicotomy can be considered in the treatment of bilateral pulmonary tuberculosis only when bilateral pneumothorax has been tried. Real damage can be avoided only if one observes certain indications, especially if the diaphragm rises very high after the first phrenicotomy. This method should be tried only in cases in which artificial pneumothorax fails. The failure of the latter usually implies the presence of adhesions which hold the diaphragm in relative fixation. There is the possibility, however, that, in phrenicotomy after bilateral pneumothorax has failed, the diaphragm may rise unduly high. This contraindicates phrenicotomy on the contralateral side. The extent of the bilateral disease is also an important indication. Recourse to operation should be had only in severe grades of disease, but disease throughout both lungs contraindicates the operation.—*Doppelseitige Phrenikusexairese bei Lungentuberkulose, L. Dünner, Deutsche med. Wchnschr., November 15, 1929, lv, 1928.*—(*H. S. W.*)

Indications for Artificial Pneumothorax and Phrenicotomy.—The combined use of pneumothorax and phrenicotomy is ill-advised. In the first place the appearance of exudates in the presence of an injured diaphragm is common. Each of these procedures has its own set of indications and these make it difficult to approach a given patient with the idea of doing both things.—*Die besonderen Indikationen des Pneumothorax und der Phrenikusausschaltung, H. Alexander, Deutsche med. Wchnschr., March 28, 1930, lvi, 518.*—(*H. S. W.*)

New Sign in Artificial Pneumothorax.—At the end of inspiration, a loud, dry, sonorous and rubbing sound was heard in the intercapsular-vertebral region of the collapsed side. It sounded as though a solid body rubbed against the internal surface of the thoracic parietes. It was most marked for two days following a pneumothorax refill. Then it would disappear and not be audible again until the next treatment. With the patient lying in the dorsal decubitus position, the sound vanished. A palpable thrill was perceived simultaneously with the appearance of the sound. The author observed this phenomenon in 3 patients receiving pneumothorax therapy. It is probably due to an "air ball" in the pleural cavity and the friction of a portion of the collapsed lung against the chest-wall.—*Su un nuovo fenomeno ascoltatorio nel pneumotorace artificiale, R. Lusena, Tubercolosi, March, 1929, xxi, 117.*—(*U. E. Z.*)

Adhesive Pleuritis in Artificial Pneumothorax.—Roentgenographs of partial pneumothorax were demonstrated. It seems that the pneumothorax pocket is not uniformly transparent to the rays but is masked by large gray areas, slightly opaque, entirely homogeneous, and quite extensive, in certain films covering almost the entire pneumothorax. The inner margin often sends prolongations toward the lung and mediastinum. The lung is ordinarily compressed against the mediastinum by the high intrapleural pressure which is maintained and its borders are not clearly defined; however the shadows are too homogeneous to be explained by a reflection of part of the pulmonary parenchyma against the thoracic wall, while on the other hand they are too extensive and continuous to be attributed to adhesions in the pleural cavity. Their location is usually more toward the lateral chest-wall and their uniform opacity recalls the appearance of small encysted effusions which develop outside the lung after abandonment of pneumothorax. In the most typical cases, however, there exists a definite pleural exudate in the lower part of the pocket, whose more opaque shadow, with an upper horizontal level, is clearly distinguishable. Without anatomical control, it seems reasonable to assert that there is a pleural thickening, or pachypleuritis. These pictures almost always follow an exudate and suggest a gelatinous or pseudomembranous extension of the same along the walls of the pneumothorax. According to the work of Mantoux and Maingot, this pachypleuritis, to be perceptible on fluoroscopic examination, must reach a thickness of at least 10 to 15 mm. For this reason, doubtless, it was demonstrable in only 6 per cent of partial pneumothorax cases examined, eliminating all doubtful or indistinct shadows. These pleural modifications are very durable and stable, and, at least in cases in whom pneumothorax is continued, may last for years. Such pleural thickening does not necessarily result in symphysis, which can usually be prevented by frequently renewed insufflations of air or nitrogen under strongly positive pressure. Partial pneumothoraces may even be enlarged by insufflating every week or several times a week with the maximum pressures available with the Küss apparatus. This procedure necessitates frequent punctures and is hardly practicable outside of a hospital or sanatorium. Even in a sanatorium, gomenolated paraffin injections may be preferable, especially for chronic pleural tuberculosis, pyopneumothorax with persistent subfebrile state, or double simultaneous pneumothorax threatened by symphysis. But insufflation has the advantage over oleo-

thorax of allowing the state of the pleural pocket and subjacent lung to be followed. It exposes less to grave complications, especially pleural ones, is easier and takes less time. That is why, in the beginning, in most cases of early symphysis or partial pneumothorax, an attempt is first made to maintain or improve the collapse by repeated insufflations under strongly positive pressure. Later, when the pneumothorax walls are become sclerotic, firmer and less permeable and the tendency to symphysis is reduced, there is less hesitancy in having recourse to paraffin injections.—*Pachypleurite au cours du pneumothorax artificiel et traitement de la symphyse pleurale, E. Bac and L. Reumaux, Section d'études scientifiques de l'Oeuvre de la Tuberculose, November 10, 1928, reported in Rev. d. l. Tuberc., February, 1929, x, 125.*—(*A. P.*)

Exudates in Artificial Pneumothorax.—Guinard first cites four personal observations among 331 pneumothorax cases at the Sanatorium de Fontenay, at Bligny, from February, 1921, to February, 1929, examples of secondary infection not due to initial pleuropulmonary perforation. Three of these patients died, one after a simple pleurotomy, another with a streptococcic infection and foetid pus, after lavage with serum and injection of gomenolated oil. The patient who recovered received pleural lavage with serum, having previously shown gram-positive diplococci in a thick purulent slightly odorous liquid. These four were the only frankly purulent cases; however, there were altogether 86 with exudate requiring puncture, 57 being frankly serous and 25 "puriform" or semipurulent. Altogether 232, or 69 per cent, showed some fluid fluoroscopically, while 99, or 31 per cent, never showed any fluid. In 89 cases, or 26 per cent, the fluid was minimal in quantity; in 57, or 18 per cent, it covered the hemidiaphragm or reached the angle of the scapula, without necessitating puncture, while 86, as stated, or 25 per cent, did require evacuation. *Relation between Technique and Exudate-Formation:* During the first four years of this period the number requiring puncture was 33 per cent, during the last four 20 per cent. The diminution is attributed to (1) shorter spacing of refills, without any modification of the terminal pressures; (2) avoiding rigorously any damage to the lung by the needle; and (3) avoiding, especially at first, any distention of adhesions or dislocation of the mediastinum. The usual intervals between refills have been 8, 10, 12 and 15 days, in cases with a "supple" pneumothorax. The rapidity of insufflation, except perhaps in the very beginning, has appeared to exert no influence on exudate-formation and all refills are carried out under pressure. All four cases of infection occurred during the earlier period, when the siphonage system of removal, usually through a trocar, was in use. Later on a closed system was employed and ordinary insufflation needles used. It was observed that when siphonage with air contact was used some small gas bubbles would appear and flow backward in the evacuating tube. These are apt to be contaminated by passage through the long rubber tubing. This condition is readily remedied by the use of Potain's aspirating apparatus. *Conclusions: 1:* Secondary pleural infection occurred in only 1.2 per cent of 331 pneumothorax cases observed over eight years. It was thought to be due to external contamination during aspiration and remedied by using a closed suction system (of Potain). *2:* Frank pleural reactions occurred in 43 per cent, 25 per cent requiring evacuation of fluid. *3:* Rapidity of insufflation seemed to have no influence but their number was reduced by (1) shorter intervals between refills, (2) care in avoiding puncture of the lung, and (3) avoidance of distention of adhesions and overpressure. In discussing the paper, Pissavy disagreed as to the innocuousness of rapid refills and emphasized the importance of another factor, the character of the pulmonary lesion, collapse of very active lesions being very apt to be complicated by exudate, as compared with indolent or slightly active lesions.—*Quelques constatations à propos des épanchements du pneumothorax, U. Guinard, Section d'études scientifiques de l'Oeuvre de la Tuberculose, February 9, 1929, reported in Rev. d. l. Tuberc., April, 1929, x, 265.*—(*A. P.*)

Pleurisy Complicating Artificial Pneumothorax.—Pleurisy in the tuberculous often remains clinically silent and unmanifested. Artificial pneumothorax is occasionally its activator. This complication occurs in 50 per cent of the patients receiving artificial-pneumothorax therapy. Some authors believe that the pleurisy is tuberculous; others ascribe its origin to an exaggerated endopleural pressure. Nonsterile gas or needles may cause pleural infection. Pleural reactions may follow a too rapid introduction of gas. In most cases the occurrence of pleurisy during pneumothorax therapy is directly attributable to carelessness in technique. By proper preparation and exercising ordinary caution much of the inconvenience to the patient and worry to the operator can be avoided.—*Per evitare la pleurite pneumotoraccica A. Rabino, Tubercolosi, November, 1928, xx, 444.*—(*U. E. Z.*)

Contralateral Pleurisy in Artificial Pneumothorax.—Hinault could find only 27 published cases, not including the five which he reports. The incidence of this complication was 1.31 per cent at the Loomis Sanatorium (Peters) and Hinault's 5 observations recorded among 418 pneumothorax cases give a proportion of 1.2 per cent, almost identical with Peters. It is interesting to note that among these 418 cases were 142 men and that all 5 pleurisies occurred in men. Altogether, serofibrinous pleurisy is unusual in the course of manifest pulmonary tuberculosis. Bezançon and Weil saw only 4 cases in six years in their specialized service, while in the general medical service there were 136 cases in this period. It is particularly to discuss the prognosis and treatment and determine the relation to subjacent pulmonary lesions that these cases are reported. The cases are summarized thus: (1) left pneumothorax, right localization, right pleurisy, fluid on the left; (2) right pneumothorax, left pleurisy, left pulmonary localization, fluid on the right; (3) left pneumothorax, pulmonary perforation, right pulmonary localization, right pleurisy, meningitis; (4) right pneumothorax, left pleurisy, fluid on the right; (5) right pneumothorax, fluid on the right, left pleurisy, pulmonary perforation. The outcome of the 5 cases was as follows: One case was completely cured, with total reëxpansion of the parenchyma of the pneumothorax side and roentgenographic integrity of both sides four years later. Two patients died in whom a bad prognosis was evident by reason of contralateral parenchymal localization preceding the pleurisy and persistent bacillary sputum. There was also a death from meningitis and one from pulmonary perforation. The immediate effects are not generally very grave and the treatment, as set forth by Mlle. Huguet, consists in decompression and repeated punctures. The later results, on the other hand, appear to be less favorable, as corroborated by Pisoni and by Peters. In two cases a bilateral pneumothorax might have been of benefit but at this period was not currently practised. According to Ameuille, any subject who develops a pleurisy has 15 chances in 100 of later developing parenchymatous pulmonary tuberculosis. Here, however, there already exists a pulmonary tuberculosis, which the pneumothorax may have arrested but which has been propagated, probably by the lymphatic route, to the opposite pleura and the lung is threatened inasmuch as the pleurisy represents a stage in the development of tuberculous lesions. Should a double pneumothorax be induced in the absence of demonstrable pulmonary localization? This seems scarcely justifiable in view of the risks and complications. It seems wiser to reserve double pneumothorax for cases with pulmonary involvement preceding the pleurisy.—*Les pleurisies contralatérales au cours du pneumothorax, V. Hinault, Section d'études scientifiques de l'Oeuvre de la Tuberculose, December 8, 1928, reported in Rev. d. l. Tuberc., February, 1929, x, 148.*—(*A. P.*)

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INDEX OF ABSTRACTS OF TUBERCULOSIS

Abdominal lymph nodes, Calcified, 35
Abrasive industry, Dust hazards in, 68
Abscess of lung, 74
——, pulmonary, Treatment of, with artificial pneumothorax, 75
Abscesses, BCG, pus from, Bacteriology of, 102
——, pulmonary, deepseated, Treatment of, 75
——, Reaction of pus in, 2
Abuses of ultraviolet radiation, Value and, 61
Acid-fast bacteria, Pigmentation of, 11
Action of nonspecific irritants on blood in tuberculosis, 66
—— —— salt-water baths, 47
Activity of tuberculosis, Diagnosis of, 21
Acute respiratory infections and pulmonary tuberculosis, 31
—— tuberculosis, Insidious and, 18
Adams, L. J. Tuberculosis of aorta, 33
Addison's disease, Pathological anatomy of, 117
Adenitis, axillary, tuberculous, Tumor of breast with, 31
Adhesive pleuritis in artificial pneumothorax, 126
Aetiology of silicosis, 68
Africa, East, interior, Tuberculosis in, 92
After-care of tuberculosis patients, 47, 120
Aged, Pleurisy in, 33
Agglutination, Cholesterol, reaction in tuberculosis, 110
Aids, Diagnostic, in pulmonary tuberculosis, 20
Air embolism in artificial pneumothorax, 58
Air-pressure, Diminished, and experimental tuberculosis, 3
Albee operation in tuberculous spondylitis, 39
Albumin and globulin, Serum, in tuberculosis, 2
Alexander, H. Indications for artificial pneumothorax and phrenicotomy, 126
Alexander, H. L., Dowell, D., and Kountz, W. B. Emphysema and heart, 75
Alexander, J. Closed drainage of empyema, 76
Allergy due to cold, 83
—— in tuberculosis, 14, 15
—— to nonhaemolytic streptococci, 83
——, Tuberculin, and antituberculosis immunity, 109
Allison, R. G. Syphilis of lung, 81
Amnès. Treatment of surgical tuberculosis by Finikoff's method, 117
Anaesthesia for thoracoplasty, 125
Anaphylactic shock, 84
Anaphylaxis with carbohydrate antigen of tubercle bacillus, 108
Anatomy, Pathological, of Addison's disease, 117
Angove, H. S., and Douthwaite, A. H. Exercises in pulmonary diseases, 120
Animal inoculation in diagnosis of surgical tuberculosis, 37
—— ——, Rapid diagnosis of tuberculosis by, 114
Antagonism between tuberculosis and poliomyelitis, 84
—— —— tumors and tuberculosis, 91
Anthracosis and tuberculosis, 68
Antibodies in experimental tuberculosis, 13
Antigen, carbohydrate, of tubercle bacillus, Anaphylaxis with, 108
——, Klopstock-Neuberg, 115.
Antigenic substances of tubercle bacillus, 12, 13
Antituberculosis immunity, Tuberculin allergy and, 109
Aorta, Tuberculosis of, 33
Appearance of symptons of tuberculosis, 17
Armand-Delille, P., and Lestocquoy, C. Diagnosis of tuberculosis of tracheobronchial lymph nodes, 94
Arnstein, A., and Huppert, M. Pleural effusions, 32

Arterial and venous blood in pulmonary tuberculosis, Oxygen and carbon dioxide of, 3
—— thrombosis, cerebral, Miliary tuberculosis with, 119
Artificial pneumothorax, 48
—— ——, Adhesive pleuritis in, 126
—— ——, Air embolism in, 58
—— —— and phrenicotomy, Indications for, 126
—— ——, Bilateral, 58
—— ——, ——, and phrenicotomy, 58
—— ——, complications of, Pleural, 52
—— ——, Effusions in, 53
—— ——, Exudates in, 127
—— ——, —— of, Treatment of, 54
—— —— in diagnosis of cavity, 28
—— —— —— differential diagnosis of pulmonary tumor and tuberculosis, 28
—— ——, Pleurisy complicating, 127
—— ——, —— in, Contralateral, 128
—— ——, sign in, New, 126
—— ——, Spontaneous after, 58
—— ——, Temperature of gas in, 54
—— ——, Treatment of pulmonary abscess with, 75
Asbestos fibres in pulmonary lesions, 69
Asbestosis, 69
Ashbury, H. E. Massive collapse of lung, 70
Aspiration in empyema, 75
—— pneumonia, Sputum flora in, 11
—— ——, X-ray image of, 28
Associated with emphysema, Pneumothorax, 77
Association of surgical and intrathoracic tuberculosis, 36
Asthma, 82, 83
Atelectasis, Physiology of, 69
Atrophy of testicles in pulmonary tuberculosis, 42
Auld, A. Asthma, 83
Axillary adenitis, tuberculous, Tumor of breast with, 31
Ayerza's disease, 81

Bac, E., and Reumaux, L. Adhesive pleuritis in artificial pneumothorax, 126
Bacilli, tubercle, Counting of, 4
——, ——, Cultivation of, 23
Bacilli, tubercle, Cultivation of, from human sputum, 23
——, ——, Demonstration of, in histological sections, 22
——, ——, Diagnostic culture of, 22
——, ——, Effect of dyes on, 10
——, ——, Fate of, in reinfected rabbits, 106
——, ——, in faeces, 107
——, ——, —— latent tuberculosis, 11
——, ——, Microscopic determination of, 22
——, ——, saponified, Immunization with, 14
——, ——, virulence of, Determination of, 10
Bacillus, paralysis-producing, Cultural and staining reactions of, 105
Bacillus, tubercle, Antigenic substances of, 12, 13
——, ——, Biology of, 5
——, ——, carbohydrate antigen of, Anaphylaxis with, 108
——, ——, Culture tube for, 3
——, ——, Cytology and microchemistry of, 4
——, ——, Filterability of, 9
——, ——, Filterable forms of, 7
——, ——, Invisible forms of, 6
——, ——, Paralytic disease in guinea pigs due to, 104
——, ——, Variability of, 5
Bacteria, acid-fast, Pigmentation of, 11
Bacteriology of pus from BCG abscesses, 102
Baer, G. Artificial pneumothorax, 48
Ball, H. A., and Evans, N. Coccidioidal granuloma, 79
Ballon, H. C. Fibrin body in pneumothorax, 77
Barker, N. W. Pathological anatomy of Addison's disease, 117
Barrett, C. D., Hayhurst, E. R., Kindel, D. J., and Neiswander, B. E. Tuberculosis and silicosis, 68
Basic feeding in tuberculosis, 46
Baths, salt-water, Action of, 47
Baum, F. Influenza and latent pulmonary tuberculosis, 67
Baylis, A. B., and MacNeal, W. J. Culture tube for tubercle bacillus, 3

BCG, 101
—— abscesses, pus from, Bacteriology of, 102
——, History of, 97
——, Immunization with, 99
—— in Belgium, Use of, 101
Bedford, D. E., and Joules, H. Bilateral spontaneous pneumothorax, 33
Beer, E. Renal tuberculosis, 41
Behnke, K. Complement fixation in tuberculosis, 24
Belgium, BCG in, Use of, 101
——, Tuberculosis mortality in, 92
Benign tuberculous spondylitis, 38
Benzyl cinnamic ester in tuberculous laryngitis, 30
v. Bergmann, G. Treatment of tuberculosis with ergosterol, 62
Berlin, A. J. Temperature of gas in artificial pneumothorax, 54
Bernstein, A. Pulmonary lymphogranulomatosis, 79
Besredka's reaction in practical work, 25
Bigler, J. A. Interpretation of roentgenograms of chest in children, 95
Bilateral artificial pneumothorax, 58
—— —— —— and phrenicotomy, 58
—— phrenicotomy, 126
—— spontaneous pneumothorax, 33
Biology of tubercle bacillus, 5
Birnbaum, G. L., and Coryllos, P. N. Size of consolidated lung in lobar pneumonia, 72
Blackfan, K. D., and Diamond, L. K. Monocytes in tuberculosis of infants and children, 96
Blair, H. C. Tuberculosis of symphysis pubis, 39
Blocking phrenic nerve, 59
Blood, Action of nonspecific irritants on, in tuberculosis, 66
——, arterial and venous, in pulmonary tuberculosis, Oxygen and carbon dioxide of, 3
Blumenberg, W., and Wiening, H. Counting of tubercle bacilli, 4
Bock, M. Mesenteric lymph-node tuberculosis, 35
Body, Fibrin, in pneumothorax, 77
——, Foreign in bronchus, 73
Boland, F. K. Penetrating chest wounds, 67
Bones, Roentgenology of, 28
Borock, M., and Wildre, D. Air embolism in artificial pneumothorax, 58
Bouessée, F., and Torin, J. Oleothorax, 54
Boyes, C. R., and White, J. S. Codliver oil in tuberculosis, 63
Braude, J. Tuberculosis of uterine adnexa, 43
Braun, R. Action of nonspecific irritants on blood in tuberculosis, 66
Breast, Tumor of, with tuberculous axillary adenitis, 31
Breathing, Rapid, from pulmonary congestion and oedema, 78
Bredow. Treatment of pulmonary abscess with artificial pneumothorax, 75
Brennemann, J., and McEmery, E. T. Aspiration in empyema, 75
Breton, A., and Pierret, R. Vernes test in tuberculosis, 25
Bretschneider, A. Action of salt-water baths, 47
Bronchiectasis, 73
——, Cavities and, 74
—— in childhood, 74
Bronchitis, Chronic, 73
Bronchography, Technique of, 27
Bronchopulmonary disease, Fluid level in, 27
Bronchoscopic findings in lobar pneumonia, 72
Bronchosinusitis disease, 67
Bronchus, Foreign body in, 73
Browder, J. Spontaneous nontuberculous pneumothorax, 77
Brown, G. E., and Horton, B. T. Allergy due to cold, 83
Brown, G. T. Cottonseed sensitization, 84
Brucella abortus, 84
Bruit d'Airain, 112
Budd, S. W., and Williams, C. Tuberculosis of thyroid, 32
Bullowa, J. G. M. Serum treatment of pneumonia, 73
Burman, Sergent and Rist. Sudden death following phrenicectomy in diabetic, 125
Burrell, L. S. T., Hare, Dorothy C., and Ross, Joan M. Tuberculous polyserositis and pericarditis, 33

Butter, Codliver oil and, in tuberculosis of mesenteric lymph nodes, 35
Buzzard, F. Tuberculosis of nervous system, 40

Calcified abdominal lymph nodes, 35
Calcium exchange in pulmonary tuberculosis, 111
Caldwell, D. W., and King, M. J. Brucella abortus, 84
Caldwell, G. W., and Dennett, R. H. Solarization of vitaglass, 62
Calmette, A. Filterable forms of tubercle bacillus, 7
——, ——. Tuberculin allergy and antituberculosis immunity, 109
——, ——., and Guérin, C. BCG, 101
——, ——., Valtis, J., and Saenz, A. Filterable elements of tuberculosis virus, 9
Cambrelin. Primary tuberculosis of larynx, 115
Campbell, H. P. Important complications of pulmonary tuberculosis, 28
Cancer of lung, 86
——, Treatment of, with tuberculin, 87
Capuani, G. F. Klopstock-Neuberg antigen, 115
Carbohydrate antigen of tubercle bacillus, Anaphylaxis with, 108
—— metabolism in tuberculosis, 1
Carbon dioxide of arterial and venous blood in pulmonary tuberculosis, Oxygen and, 3
Carcinoma of lungs, Primary, 89
Carcinomatosis, Fresh tuberculous foci and, 91
Case of inoculation lupus, 44
Caussimon, J. Hilar reactions and pulmonary tuberculosis, 104
Cauterization, Thoracoscopy and, 60
Cavities and bronchiectasis, 74
——, Prognosis of, 92
Cavity, diagnosis of, Artificial pneumothorax in, 28
Cellular reactions in tuberculosis, 103
Cement dust lung, 68
Cerebral arterial thrombosis, Miliary tuberculosis with, 119
Cerebrospinal fluid, Tuberculous meningitis with unusual, 40
Chandler, F. G. Conditions simulating tuberculosis, 22
Chandler, F. G. New thoracoscope, 125
——, ——. ——. Puncture of chest, 60
Chandler, G. J., and Holmes, E. B. X-ray findings of heart, 82
Chest, Puncture of, 60
——, roentgenograms of, in children, Interpretation of, 95
—— wounds, Penetrating, 67
Childhood, Bronchiectasis in, 74
Children, hospitalized, in Iowa, Tuberculin reactions among, 95
——, roentgenograms of chest in, Interpretation of, 95
——, tuberculosis of infants and, Monocytes in, 96
Cholesterol agglutination reaction in tuberculosis, 110
—— and resistance in pulmonary tuberculosis, 1
Chronic bronchitis, 73
—— febrile conditions without organic findings, 85
Churchill, E. D., and Cope, O. Rapid breathing from pulmonary congestion and oedema, 78
Cicconardi, G. Spontaneous after artificial pneumothorax, 58
Cinnamic ester, Benzyl, in tuberculous laryngitis, 30
Circulation in tuberculosis, 2
Clark, W. I. Dust hazards in abrasive industry, 68
Clauberg, K. W. Immunization with saponified tubercle bacilli, 14
——, ——. ——., and Lange, L. Filterabiliy of tubercle bacillus, 9
Climate and lupus vulgaris, 44
Closed drainage of empyema, 76
Coblentz, W. W. Sources of ultraviolet radiation, 62
Coccidioidal granuloma, 79
—— —— and tuberculosis, 80
Codliver oil and butter in tuberculosis of mesenteric lymph nodes, 35
—— —— in tuberculosis, 63
Coincident malignant tumor and tuberculosis, 87
Cold, Allergy due to, 83
Collapse, Massive, of lung, 69, 70
——, ——, Postoperative, and pneumonia, 70

Collapse, Massive, Postoperative, Treatment of, 71
——, ——, X-ray features of, 71
—— therapy in tuberculosis, 49
Colloid lability, Effect of complications on, 1
Combined tuberculin and gold treatment of tuberculosis, 65
Comparison of tuberculous with other infections, 29
Complement fixation in tuberculosis, 24
Complicating artificial pneumothorax, Pleurisy, 127
Complications, Effect of, on colloid lability, 1
—— of artificial pneumothorax, Pleural, 52
—— —— pulmonary tuberculosis, Important, 28
Conditions simulating tuberculosis, 22
Congenital cystic lung, 80
Congestion, pulmonary, and oedema, Rapid breathing from, 78
Consolidated lung, Size of, in lobar pneumonia, 72
Contralateral pleurisy in artificial pneumothorax, 128
Cope, O., and Churchill, E. D. Rapid breathing from pulmonary congestion and oedema, 78
Copper, Treatment of tuberculosis with, 65
Corper, H. J., and Uyei, N. Diagnostic culture of tubercle bacilli, 22
Coryllos, P. N. Bronchoscopic findings in lobar pneumonia, 72
——, ——. ——. Postoperative massive collapse and pneumonia, 70
——, ——. ——., and Birnbaum, G. L. Size of consolidated lung in lobar pneumonia, 72
Costa reaction, 25
Cottonseed sensitization, 84
Counting of tubercle bacilli, 4
Crandall, L. A., and Walsh, E. L. Effect of iodized oils on serous membranes, 65
Crisis, Haemoclastic, and tuberculin, 114
Cultivation of tubercle bacilli, 23
—— —— —— —— from human sputum, 23
Cultural and staining reactions of paralysis-producing bacillus, 105
Culture, Diagnostic, of tubercle bacilli, 22
—— method of Hahn, 23
Culture tube for tubercle bacillus, 3
Cummins, W. T., Smith, J. K., and Halliday, C. H. Coccidioidal granuloma and tuberculosis, 80
Curettage in tuberculous epididymitis, 42
Cystic lung, Congenital, 80
Cytology and microchemistry of tubercle bacillus, 4

Davidson, M. Intrathoracic tumors, 85
Day, H. F., Sisson, W. R., and Vogt, E. C. Anaphylactic shock, 84
Death, Sudden, following phrenicectomy in diabetic, 125
Deepseated pulmonary abscesses, Treatment of, 75
Del Rio, S. Diminished air-pressure and experimental tuberculosis, 3
Demonstration of tubercle bacilli in histological sections, 22
Denk, W. Blocking phrenic nerve, 59
Dennett, R. H., and Caldwell, G. W. Solarization of vitaglass, 62
Derick, C. L., and Swift, H. M. Allergy to nonhaemolytic streptococci, 83
Determination of virulence of tubercle bacilli, 10
Deutsch, E. Prognosis of tuberculosis of joints, 37
Diabetic, phrenicectomy in, Sudden death following, 125
Diagnose tuberculosis, Failure to, 22
Diagnosis, differential, of pulmonary tumor and tuberculosis, Artificial pneumothorax in, 28
——, Early, of pulmonary tuberculosis, 20
——, ——, Symptoms and signs in, 20
—— of activity of tuberculosis, 21
—— —— cavity, Artificial pneumothorax in, 28
—— —— pulmonary tuberculosis, Early, 20
—— —— sinus disease, 67
—— —— surgical tuberculosis, Animal inoculation in, 37
—— —— tuberculosis by animal inoculation, Rapid, 114
—— —— —— of tracheobronchial lymph nodes, 94
——, X-ray, of intrathoracic tumors, 87

Diagnosis, X-ray, "quality," in pulmonary tuberculosis, 26
Diagnostic aids in pulmonary tuberculosis, 20
—— culture of tubercle bacilli, 22
—— difficulties in tuberculous meningitis, 119
—— errors in pulmonary tuberculosis, 21
Diamond, L. K., and Blackfan, K. D. Monocytes in tuberculosis of infants and children, 96
Diaphragmatic pleurisy, 80
Diathesis, Haemorrhagic, in tuberculosis, 103
Dickey, L. B. Primary tuberculous infection of tonsil, 115
——, ——. ——. Size of intracutaneous tuberculin reactions, 95
Dickinson, W. H. Observations on tuberculosis, 22
Dienes, L. Antigenic substances of tubercle bacillus, 12, 13
——, ——. Tuberculin type of sensitization with egg-white, 107
Diet in tuberculosis, 46
——, Salt-free, in pulmonary tuberculosis, 47
Dietrich, H. Nursemaids and teachers as sources of tuberculous infection, 93
Differential diagnosis of pulmonary tumor and tuberculosis, Artificial pneumothorax in, 28
Difficulties, Diagnostic, in tuberculous meningitis, 119
Diminished air-pressure and experimental tuberculosis, 3
Disease, Addison's, Pathological anatomy of, 117
——, Ayerza's, 81
——, bronchopulmonary, Fluid level in, 27
——, Bronchosinusitis, 67
——, sinus, Diagnosis of, 67
Diseases, pulmonary, Exercises in, 120
Dispensary treatment of tuberculosis, 46
Dittrich, O. Primary tuberculosis of skin, 43
Dix-Perkin, A. J., and McLaughlin. A. I. G. Bruit d'Airain, 112
Dölter, W., and Keller, W. Sensitiveness to glycerin-bouillon, 17
Doria, R. Red-cell sedimentation reaction, 115
Dorn, E. Perspiration test for tuberculosis, 28
Douthwaite, A. H., and Angove, H. S. Exercises in pulmonary diseases, 120
Dowell, D., Alexander, H. L., and Kountz, W. B. Emphysema and heart, 75
Drainage of empyema, Closed, 76
Dresser, R. Foreign body in bronchus, 73
Dumarest, F. Immediate risks of thoracoplasty, 125
Dünner, L. Bilateral phrenicotomy, 126
——, ——., and Heilborn. Bilateral artificial pneumothorax and phrenicotomy, 58
Dust, Cement, lung, 68
—— hazards in abrasive industry, 68
—— inhalation, Treatment of pulmonary tuberculosis by, 47
Dyes, Effect of, on tubercle bacilli, 10

Early diagnosis of pulmonary tuberculosis, 20
—— ——, Symptoms and signs in, 20
—— pulmonary tuberculosis, Sign of, 19
Earp, J. R. Heliotherapy, 60
East Africa, interior, Tuberculosis in, 92
Edema. See oedema.
Effect of complications on colloid lability, 1
—— —— dyes on tubercle bacilli, 10
—— —— fermentation on tuberculin, 16
—— —— iodized oils on serous membranes, 65
Effusions in artificial pneumothorax, 53
——, Pleural, 32
——, Pleuritic, 32
Egg-white, Tuberculin type of sensitization with, 107
Embolism, Air, in artificial pneumothorax, 58
Emphysema and heart, 75
——, Pneumothorax associated with, 77
Empyema, Aspiration in, 75
——, drainage of, Closed, 76
——, Pneumococcic, 75
——, Treatment of, 76
Enders, J. T. Anaphylaxis with carbohydrate antigen of tubercle bacillus, 108
Environment, tuberculous, Infant mortality in, 102
Epididymitis, tuberculous, Curettage in, 42

Ergosterol, Treatment of tuberculosis with, 62
Ernlund, C. B. Laryngeal tuberculosis, 30
Ernst, F. Treatment of pulmonary tuberculosis with solganol, 64
——, ——., and Rickmann, L. Prognostic tests, 92
Errors, Diagnostic, in pulmonary tuberculosis, 21
Esophagus. See oesophagus.
Etiology. See aetiology.
Evans, N., and Ball, H. A. Coccidioidal granuloma, 79
Exchange, Calcium, in pulmonary tuberculosis, 111
Exercises in pulmonary diseases, 120
Experimental tuberculosis, Antibodies in, 13
—— ——, Diminished air-pressure and, 3
Exudates in artificial pneumothorax, 127
—— of artificial pneumothorax, Treatment of, 54

Factors in recovery from tuberculosis, 45
Faeces, Tubercle bacilli in, 107
Failure to diagnose tuberculosis, 22
Farrell, J. T. X-ray diagnosis of intrathoracic tumors, 87
Fate of tubercle bacilli in reinfected rabbits, 106
Febrile conditions, Chronic, without organic findings, 85
Feeding in tuberculosis, Basic, 46
Fermentation, Effect of, on tuberculin, 16
Ferments, proteolytic, Serum, in pulmonary tuberculosis, 1
Fernier, L., Saleur, H., and Parisot, J. Bacteriology of pus from BCG abscesses, 102
Fertik, I., and Pick, M. Codliver oil and butter in tuberculosis of mesenteric lymph nodes, 35
Fibres, Asbestos, in pulmonary lesions, 69
Fibrin body in pneumothorax, 77
Fibrosarcoma of mediastinum, 91
Figley, K. D. Asthma, 82
Filter, T. Tuberculosis and syphilis, 81
Filterability of tubercle bacillus, 9
—— —— tuberculosis virus, 6
Filterable elements of tuberculosis virus, 9
Filterable forms of tubercle bacillus, 7
Findings, Bronchoscopic, in lobar pneumonia, 72
——, X-ray, of heart, 82
Finikoff's method, Treatment of surgical tuberculosis by, 117
Finkel-Karpovsky, A. Sputum flora in aspiration pneumonia, 11
Fishberg, M. Cancer of lung, 86
Flocculation test, Serum, 25
Flora, Sputum, in aspiration pneumonia, 11
Fluid, cerebrospinal, Tuberculous meningitis with unusual, 40
—— level in bronchopulmonary disease, 27
Fluids, limitation of, Treatment of tuberculosis by, 47
Foci, tuberculous, Fresh, and carcinomatosis, 91
Following phrenicectomy in diabetic, Sudden death, 125
Fontaine, J. Results of oleothorax, 57
Ford, A. P. Dispensary treatment of tuberculosis, 46
Foreign body in bronchus, 73
Forms of tubercle bacillus, Filterable, 7
—— —— —— ——, Invisible, 6
Fortune, C. H. Coincident malignant tumor and tuberculosis, 87
Frederiksen, J. A. Metallic salt therapy, 63
Fresh tuberculous foci and carcinomatosis, 91
Freund, J. Sensitiveness of guinea pigs to tuberculin, 109
Fried, B. M. Primary carcinoma of lungs, 89
Friedrich, H., and Knorr, M. Animal inoculation in diagnosis of surgical tuberculosis, 37
Fungi, Pathogenic, 82

Gainsborough, H., and Jory, P. J. Benzyl cinnamic ester in tuberculous laryngitis, 30
Gardner, L. L., St. John, J. H., and Simmons, J. S. Nematode infestation of lung, 79
Gas, Subdiaphragmatic, 81
——, Temperature of, in artificial pneumothorax, 54

Genitourinary tuberculosis, Physiotherapy of, 117
Gerling, W. Case of inoculation lupus, 44
Gibson, D. N. Congenital cystic lung, 80
Giercke, H. W. Treatment of pulmonary tuberculosis with metallosan, 64
Ginsberg, R., and Pewsner, R. Carbohydrate metabolism in tuberculosis, 1
Globulin, Serum albumin and, in tuberculosis, 2
Gloyne, S. R. Asbestos fibres in pulmonary lesions, 69
Glycerin-bouillon, Sensitiveness to, 17
Gold treatment of tuberculosis, Combined tuberculin and, 65
Goldberg, B. Newer treatment of tuberculosis, 119
——, ——. Uses and limitations of sanatoria, 45
Golden, R., and Reeves, R. I. Calcified abdominal lymph nodes, 35
Gorter, E. Diagnosis of activity of tuberculosis, 21
Granuloma, Coccidioidal, 79
——, ——, and tuberculosis, 80
Griffin, W. A. Intestinal tuberculosis, 34
Groedel, F. M., and Wachter, R. X-ray "quality" diagnosis in pulmonary tuberculosis, 26
Gross, R., and Lasch, F. Serum proteolytic ferments in pulmonary tuberculosis, 1
Grumbach, A., and Werner, A. J. Besredka's reaction in practical work, 25
Guérin. History of BCG, 97
Guérin, C., and Calmette, A. BCG, 101
Guinard, U. Exudates in artificial pneumothorax, 127
——, ——. Fluid level in bronchopulmonary disease, 27
Guinea pigs, Paralytic disease in, due to tubercle bacillus, 104
—— ——, Sensitiveness of, to tuberculin, 109
Gutmann, M. J. Results of tuberculin therapy, 66

Haemoclastic crisis and tuberculin, 114
Haemoptyses, tuberculous, Hereditary, 17
Haemoptysis, Pneumothorax or transfusion for, 54
Haemorrhagic diathesis in tuberculosis, 103
Hagedorn, K. Ziehl-Neelsen method preferred, 22
Hahn, Culture method of, 23
Hallam, R. Lupus vulgaris, 44
Halliday, C. H., Cummins, W. T., and Smith, J. K. Coccidioidal granuloma and tuberculosis, 80
Hantschmann, L., and Steube, M. Effect of complications on colloid lability, 1
Hare, Dorothy, Ross, Joan M., and Burrell, L. S. T. Tuberculous polyserositis and pericarditis, 33
Harkavy, J. Abscess of lung, 74
Hart, D. Treatment of empyema, 76
Hartung, A. Bronchiectasis, 73
Hawes, J. B., 2nd. Diet in tuberculosis, 46
Hayhurst, E. R., Kindel, D. J., Neiswander, B. E., and Barrett, C. D. Tuberculosis and silicosis, 68
Hazards, Dust, in abrasive industry, 68
Heaf, F. Sanocrysin treatment, 64
Heart, Emphysema and, 75
——, X-ray findings of, 82
Heffeman, P. Aetiology of silicosis, 68
Heilborn and Dünner, L. Bilateral artificial pneumothorax and phrenicotomy, 58
Heliotherapy, 60
Helpin, Treatment of pulmonary tuberculosis with, 66
Henderson, Y. Physiology of atelectasis, 69
Henius, K., and Wissing. Treatment of pulmonary tuberculosis with lopion, 65
Hennell, H. Massive collapse of lung, 69
Hennes, H. Treatment of pulmonary tuberculosis by dust inhalation, 47
Herbert, G. T. X-rays in pulmonary tuberculosis, 26
Hereditary tuberculous haemoptyses, 17
Hilar reactions and pulmonary tuberculosis, 104
Hilgers, W. E., and Sachs-Müke. Complement fixation in tuberculosis, 24
Hill, A. M., and Williams, L. R. Appearance of symptoms of tuberculosis, 17
——, ——. ——., —— ——, ——. ——. Diagnostic aids in pulmonary tuberculosis, 20
Hilum tuberculosis, 32

Hinault, V. Contralateral pleurisy in artificial pneumothorax, 128
Hinton, W. A., and Stuart, G. O. Cholesterol agglutination reaction in tuberculosis, 110
Hinze, V. Cholesterol and resistance in pulmonary tuberculosis, 1
Histological sections, tubercle bacilli in, Demonstration of, 22
History of BCG, 97
Hollaender, L. Diaphragmatic pleurisy, 80
Hollo, J. Incipiency of pulmonary tuberculosis, 19
Holmes, E. B., and Chandler, G. J. X-ray findings of heart, 82
Holmes, G. W. X-ray features of massive collapse, 71
Homann, E. Malignant tumors of lung, 86
——, ——. Treatment of pulmonary tuberculosis with helpin, 66
Horder, T. Pleuritic effusions, 32
Horton, B. T., and Brown, G. E. Allergy due to cold, 83
Hospitalized children in Iowa, Tuberculin reactions among, 95
Howard, W. T., Jr., Pearl, R., and Sutton, A. C. Treatment of cancer with tuberculin, 87
Howarth, W. G. Tuberculosis of larynx, 31
Hudson, B., and Maurer, G. Thoracoscopy and cauterization, 60
Human sputum, Cultivation of tubercle bacilli from, 23
Huppert, M., and Arnstein, A. Pleural effusions, 32

Immediate risks of thoracoplasty, 125
Immunity, antituberculosis, Tuberculin allergy and, 109
——, Skin as organ of, 16
——, —— tuberculosis and, 44
—— to tuberculosis, 16
Immunization with BCG, 99
—— —— saponified tubercle bacilli, 14
Important complications of pulmonary tuberculosis, 28
Incipiency of pulmonary tuberculosis, 19
Indications for artificial pneumothorax and phrenicotomy, 126
Industry, abrasive, Dust hazards in, 68
Infant mortality in tuberculous environment, 102
Infants and chilren, tuberculosis of, Monocytes in, 96
——, tuberculosis in, Milk and, 93
Infection, Mixed, in pulmonary tuberculosis, 11
——, tuberculous, of tonsil, Primary, 115
——, ——, sources of, Nursemaids and teachers as, 93
Infections, Comparison of tuberculous with other, 29
——, respiratory, Acute, and pulmonary tuberculosis, 31
Influenza and latent pulmonary tuberculosis, 67
Inhalation, dust, Treatment of pulmonary tuberculosis by, 47
Inoculation, Animal, in diagnosis of surgical tuberculosis, 37
——, ——, Rapid diagnosis of tuberculosis by, 114
—— lupus, Case of, 44
Insidious and acute tuberculosis, 18
Interior East Africa, Tuberculosis in, 92
Intermediate nitrogen metabolism, lung in, Rôle of, 3
Interpretation of roentgenograms of chest in children, 95
Intestinal tuberculosis, 34
Intracutaneous tuberculin reactions, Size of, 95
Intrathoracic tuberculosis, Association of surgical and, 36
—— tumors, 85
—— ——, diagnosis of, X-ray, 87
Invisible forms of tubercle bacillus, 6
Iodized oils, Effect of, on serous membranes, 65
Iridocyclitis, treatment of, X-ray, 29
Irritability, Vasomotor, and skin reactions, 2
Irritants, nonspecific, Action of, on blood in tuberculosis, 66
Iversen, P., and Johansen, E. H. Pleural eflusions, 32

Jagoda, F. X-ray image of aspiration pneumonia, 28
Jahn, F. Chronic febrile conditions without organic findings, 85

Jessen, H. Thoracoplasty, 52
Johansen, E. H., and Iversen, P. Pleural effusions, 32
Johnson, D. J. G. Hilum tuberculosis, 32
Joints, tuberculosis of, Prognosis of, 37
Jordan, R. Vernes test in tuberculosis, 26
Jory, P. J., and Gainsborough, H. Benzyl cinnamic ester in tuberculous laryngitis, 30
Joules, H., and Bedford, D. E. Bilateral spontaneous pneumothorax, 33

Kapeller. Microscopic determination of tubercle bacilli, 22
Karfiol, G. Treatment of tuberculosis by limitation of fluids, 47
Keller, W., and Dölter, W. Sensitiveness to glycerin-bouillon, 17
Kern, R. A., and Schenck, H. P. Diagnosis of sinus disease, 67
Kerzmann, J. A. Bilateral artificial pneumothorax, 58
Keyes, E. L. Curettage in tuberculous epididymitis, 42
Kindel, D. J., Neiswander, B. E., Barrett, C. D., and Hayhurst, E. R. Tuberculosis and silicosis, 68
King, M. J., and Caldwell, D. W. Brucella abortus, 84
Kirchner, O. Biology of tubercle bacillus, 5
——, ——. Filterable forms of tubercle bacillus, 7
Kirk, E., and Pendergrass, E. P. Subdiaphragmatic gas, 81
Kirschner, M. Albee operation in tuberculous spondylitis, 39
——, ——. Tuberculous pyonephrosis diagnosed spondylitis, 42
Klare, K. Relation of tuberculous meningitis to other forms of tuberculosis, 40
Kleine, F. K. Tuberculosis in interior East Africa, 92
Klopstock-Neuberg antigen, 115
Knasyi, G. Cytology and microchemistry of tubercle bacillus, 4
Knee and rib, Tuberculous osteitis of, 39
Knorr, M., and Friedrich, H. Animal inoculation in diagnosis of surgical tuberculosis, 37
Koldajew, B., and Kutzenok, B. Reaction of pus in abscesses, 2
Komis, A. Effect of fermentation on tuberculosis, 16
König, E. Albee operation in tuberculous spondylitis, 39
Konstam, G. L. S., and Turnbull, H. M. Ayerza's disease, 81
Kountz, W. B., Alexander, H. L., and Dowell, D. Emphysema and heart, 75
Krasso, H. Pneumothorax associated with emphysema, 77
Krassowitzkaja, S., and Tscharny, A. Rôle of lung in intermediate nitrogen metabolism, 3
Kugelmass, I. N., and Mayer, E. Basic feeding in tuberculosis, 46
Küss, G. Oleothorax, 55
Kuss, H. Surgical tuberculosis, 117
Kutzenok, B., and Koldajew, B. Reaction of pus in abscesses, 2

Lability, colloid, Effect of complications on, 1
Lamy, L. Antagonism between tuberculosis and poliomyelitis, 84
Lange, B., and Lydtin, K. Determination of virulence of tubercle bacilli, 10
Lange, L., and Clauberg, K. W. Filterability of tubercle bacillus, 9
Langstein, L. Diagnostic difficulties in tuberculous meningitis, 119
Laryngeal tuberculosis, 30
Laryngitis, tuberculous, Benzyl cinnamic ester in, 30
Larynx, Tuberculosis of, 31
——, —— ——, Primary, 115
Lasch, F., and Gross, R. Serum proteolytic ferments in pulmonary tuberculosis, 1
Latent pulmonary tuberculosis, Influenza and, 67
—— tuberculosis, Tubercle bacilli in, 11
LeBlanc. Comparison of tuberculous with other infections, 29
Ledermann, K. G. Reaction of lymph nodes to tuberculin, 16
——, ——. ——. Testicular tuberculin reaction, 17
Leopold, J. S., and Rothstein, J. L. Miliary tuberculosis with cerebral arterial thrombosis, 119

Lestocquoy, C., and Armand-Delille, P. Diagnosis of tuberculosis of tracheobronchial lymph nodes, 94
Level, Fluid, in bronchopulmonary disease, 27
Levy, Margarete. After-care of tuberculosis patients, 47
Levy, S. Primary tuberculosis of skin, 43
Lewis, P. A., and Shope, R. E. Cultural and staining reactions of paralysis-producing bacillus, 105
——, ——. ——., —— ——, ——. ——. Paralytic disease in guinea pigs due to tubercle bacillus, 104
Li, B. D., and Lindemann, M. Filterable forms of tubercle bacillus, 7
Lichtenstein, Stefania. Cultivation of tubercle bacilli, 23
Light, quartz, Treatment of pulmonary tuberculosis with, 61
Limitation of fluids, Treatment of tuberculosis by, 47
Limitations of sanatoria, Uses and, 45
Lindemann, M., and Li, B. D. Filterable forms of tubercle bacillus, 7
Linser, P. Skin tuberculosis and immunity, 44
Lipatren in pulmonary tuberculosis, 66
——, Treatment of pulmonary tuberculosis with, 66
Lipoid treatment and red-cell sedimentation reaction, 66
Liver and pancreas in tuberculosis, 34
Lobar pneumonia, Bronchoscopic findings in, 72
—— ——, Size of consolidated lung in, 72
Loewenstein, E., and Singer, E. Invisible forms of tubercle bacillus, 6
Lopion, Treatment of pulmonary tuberculosis with, 65
Lord, F. T. Acute respiratory infections and pulmonary tuberculosis, 31
Lorey, A. Technique of bronchography, 27
Lotheissen, G. Surgery of lung, 51
Lowenburg, H. Pneumococcic empyema, 75
Lucke, B., McCutcheon, M., Strumia, M., Mudd, S., and Mudd, E. B. H. Antibodies in experimental tuberculosis, 13
Lung, Abscess of, 74.
Lung, Cancer of, 86
——, Cement dust, 68
——, consolidated, Size of, in lobar pneumonia, 72
——, cystic, Congenital, 80
—— in intermediate nitrogen metabolism, Rôle of, 3
——, Massive collapse of, 69, 70
——, Nematode infestation of, 79
——, Surgery of, 51
——, Syphilis of, 81
——, tumors of, Malignant, 86
Lungs, carcinoma of, Primary, 89
——, Surgery of oesophagus and, 52
——, tuberculous, Mineral content of, 2
Lupus inoculation, Case of, 44
—— vulgaris, 44
—— ——, Climate and, 44
Lurie, M. B. Fate of tubercle bacilli in reinfected rabbits, 106
Lusena, R. New sign in artificial pneumothorax, 126
Lydtin, K., and Lange, B. Determination of virulence of tubercle bacilli, 10
Lymph nodes, abdominal, Calcified, 35
—— ——, mesenteric, tuberculosis of, Cod-liver oil and butter in, 35
—— ——, Reaction of, to tuberculin, 16
—— ——, tracheobronchial, tuberculosis of, Diagnosis of, 94
Lymphadenoma, 79
Lymph-node tuberculosis, Mesenteric, 35
Lymphogranulomatosis, Pulmonary, 79

Macchiaro, G. Haemoclastic crisis and tuberculin, 114
MacNeal, W. J., and Baylis, A. B. Culture tube for tubercle bacillus, 3
Maendl, H. Pleural pressure after phrenicectomy, 59
——, ——., and Schwarzmann, E. Phrenicoexairesis, 59
Magill, I. W. Anaesthesia for thoracoplasty, 125
Malaria, Tuberculosis and, 84
Malignant tumor and tuberculosis, Coincident, 87
—— tumors of lung, 86
Marberger, A. Failure to diagnose tuberculosis, 22

Markowicz, W. Pulmonary tuberculosis and sport, 46
Massive collapse of lung, 69, 70
—— ——, Postoperative, and pneumonia, 70
—— ——, ——, Treatment of, 71
—— ——, X-ray features of, 71
Maurer, A. Technique of thoracoplasty, 122
——, ——., and Rolland, J. Technique of thoracoplasty, 121, 124
Maurer, G., and Hudson, B. Thoracoscopy and cauterization, 60
Mayer, A. Pregnancy and tuberculosis, 85
Mayer, E., and Kugelmass, I. N. Basic feeding in tuberculosis, 46
Mazzucato, M. Tubercle bacilli in faeces, 107
McCutcheon, M., Strumia, M., Mudd, S., Mudd, E. B. H., and Lucke, B. Antibodies in experimental tuberculosis, 13
McEmery, E. T., and Brennemann, J. Aspiration in empyema, 75
McLaughlin, A. I. G., and Dix-Perkin, A. J. Bruit d'Airain, 112
Mechanism of phrenicoexairesis, 59
Mediastinum, Fibrosarcoma of, 91
Meissner, Gertrud. Effect of dyes on tubercle bacilli, 10
Melzer, E. Cavities and bronchiectasis, 74
Meningitis, 40
——, tuberculous, Diagnostic difficulties in, 119
——, ——, Relation of, to other forms of tuberculosis, 40
——, ——, with unusual cerebrospinal fluid, 40
Menon, T. K., and Menon, T. B. Lymphadenoma, 79
Menzel, R. Effusions in artificial pneumothorax, 53
Mesenteric lymph nodes, tuberculosis of, Codliver oil and butter in, 35
—— lymph-node tuberculosis, 35
Metabolism, Carbohydrate, in tuberculosis, 1
——, nitrogen, intermediate, lung in, Rôle of, 3
Metallic salt therapy, 63, 64
Metallosan, Treatment of pulmonary tuberculosis with, 64
Method, Culture, of Hahn, 23
——, Finikoff's, Treatment of surgical tuberculosis by, 117
——, Ziehl-Neelsen preferred, 22
Microchemistry, Cytology and, of tubercle bacillus, 4
Microscopic determination of tubercle bacilli, 22
Middleton, E. L. Silicosis, 68
Miliary tuberculosis, 44
—— —— with cerebral arterial thrombosis, 119
Milk and tuberculosis in infants, 93
Mineral content of tuberculous lungs, 2
Misson. Pneumothorax or transfusion for haemoptysis, 54
Mixed infection in pulmonary tuberculosis, 11
Moeller, A. Skin as organ of immunity, 16
Monceaux, R. Calcium exchange in pulmonary tuberculosis, 111
Monocytes in tuberculosis of infants and children, 96
Morosowa, E., and Platonof, G. Mixed infection in pulmonary tuberculosis, 11
Mortality, Infant, in tuberculous environment, 102
——, Tuberculosis, in Belgium, 92
Mudd, S., Mudd, E. B. H., Lucke, B., McCutcheon, M., and Strumia, M. Antibodies in experimental tuberculosis, 13
Müller, W. Treatment of pulmonary tuberculosis with tebeform, 65.
Münchbach, W. Treatment of pulmonary tuberculosis with lipatren, 66

Neiswander, B. E., Barrett, C. D., Hayhurst, E. R., and Kindel, D. J. Tuberculosis and silicosis, 68
Nematode infestation of lung, 79
Nerve, phrenic, Blocking, 59
Nervous system, Tuberculosis and, 40
Neuda, P. Soft palate in pulmonary tuberculosis, 29
New sign in artificial pneumothorax, 126
—— thoracoscope, 125
Newer treatment of tuberculosis, 119
Nitrogen metabolism, intermediate, Rôle of lung in, 3
Nonhaemolytic streptococci, Allergy to, 83

Nonspecific irritants, Action of, on blood in tuberculosis, 66
Nontuberculous pneumothorax, Spontaneous, 77
Nursemaids and teachers as sources of tuberculous infection, 93

O'Brien, E. J. Thoracic surgery, 51
O'Brien, F. E. Treatment of pulmonary tuberculosis with quartz light, 61
Observations on tuberculosis, 22
Ochsner, A. Chronic bronchitis, 73
Oedema, Rapid breathing from pulmonary congestion and, 78
Oesophagus and lungs, Surgery of, 52
Oil, Codliver, and butter in tuberculosis of mesenteric lymph nodes, 35
——, ——, in tuberculosis, 63
Oils, iodized, Effect of, on serous membranes, 65
Olbrechts, E. Treatment of exudates of artificial pneumothorax, 54
——, ——. Tuberculosis mortality in Belgium, 92
Oleothorax, 54, 55
——, Results of, 57
Operation, Albee, in tuberculous spondylitis, 39
Organ of immunity, Skin as, 16
Organic findings, Chronic febrile conditions without, 85
Orthopaedic tuberculosis, Treatment of, by vigantol, 37
Osol, A. Demonstration of tubercle bacilli in histological sections, 22
Osteitis, Tuberculous, of knee and rib, 39
Oumansky, V., and Paisseau, G. Tuberculous rheumatism, 36
Oxygen and carbon dioxide of arterial and venous blood in pulmonary tuberculosis, 3

Paisseau, G., and Oumansky, V. Tuberculous rheumatism, 36.
Palate, Soft, in pulmonary tuberculosis, 29
Pancreas in tuberculosis, Liver and, 34
Paralysis-producing bacillus, Cultural and staining reactions of, 105
Paralytic disease in guinea pigs due to tubercle bacillus, 104
Parisot, J., and Saleur, H. Infant mortality in tuberculous environment, 102
——, ——., Fernier, L., and Saleur, H. Bacteriology of pus from BCG abscesses, 102
Pathogenic fungi, 82
Pathological anatomy of Addisons' disease, 117
Patients, tuberculosis, After-care of, 47, 120
Paulsen, E. Lipoid treatment and red-cell sedimentation reaction, 66
Pearl, R., Sutton, A. C., and Howard, W. T., Jr. Treatment of cancer with tuberculin, 87
Peers, R. A. Factors in recovery from tuberculosis, 45
Pendergrass, E. P., and Kirk, E. Subdiaphragmatic gas, 81
Penetrating chest wounds, 67
Pericarditis, Tuberculous polyserositis and, 33
Perspiration test for tuberculosis, 28
Petroff, S. A. Variability of tubercle bacillus, 5
Petschacher, L. Diagnostic errors in pulmonary tuberculosis, 21
Petterson, W. Treatment of pulmonary tuberculosis with metallosan, 64
Pewsner, R., and Ginsberg, R. Carbohydrate metabolism in tuberculosis, 1
Pfannenstiel, W. Rapid diagnosis of tuberculosis by animal inoculation, 114
Phrenic nerve, Blocking, 59
Phrenicectomy in diabetic, Sudden death following, 125
——, Pleural pressure after, 59
Phrenicoexairesis, 59
——, Mechanism of, 59
Phrenicotomy, Bilateral, 126
——, —— artificial pneumothorax and, 58
——, Indications for artificial pneumothorax and, 26
Physiology of atelectasis, 69
Physiotherapy of genitourinary tuberculosis, 117
Pick, M., and Fertik, I. Codliver oil and butter in tuberculosis of mesenteric lymph nodes, 35
Pierret, R., and Breton, A. Vernes test in tuberculosis, 25

Pigmentation, 63
—— of acid-fast bacteria, 11
Plath. Treatment of surgical tuberculosis by vigantol, 37
Platonof, G., and Morosowa, E. Mixed infection in pulmonary tuberculosis, 11
Pleural complications of artificial pneumothorax, 52
—— effusions, 32
—— pressure after phrenicectomy, 59
Pleurisy complicating artificial pneumothorax, 127
——, Diaphragmatic, 80
—— in aged, 33
—— —— artificial pneumothorax, Contralateral, 128
Pleuritic effusions, 32
Pleuritis, Adhesive, in artificial pneumothorax, 126
Pneumococcic empyema, 75
Pneumonia, aspiration, Sputum flora in, 11
——, ——, X-ray image of, 28
——, lobar, Bronchoscopic findings in, 72
——, ——, Size of consolidated lung in, 72
——, Postoperative massive collapse and, 70
——, treatment of, Serum, 73
Pneumothorax, Artificial, 48
——, ——, Adhesive pleuritis in, 126
——, ——, Air embolism in, 58
——, ——, and phrenicotomy, Indications for, 126
——, ——, Bilateral, 58
——, ——, ——, and phrenicotomy, 58
——, ——, Complications of, Pleural, 52
——, ——, Effusions in, 53
——, ——, Exudates in, 127
——, ——, —— of, Treatment of, 54
——, ——, in diagnosis of cavity, 28
——, ——, —— differential diagnosis of pulmonary tumor and tuberculosis, 28
——, ——, Pleurisy complicating, 127
——, ——, —— in, Contralateral, 128
——, ——, Sign in, New, 126
——, ——, Spontaneous after, 58
——, ——, Temperature of gas in, 54
——, ——, Treatment of pulmonary abscess with, 75
Pneumothorax associated with emphysema, 77
——, Fibrin body in, 77
Pneumothorax, nontuberculous, Spontaneous, 77
—— or transfusion for haemoptysis, 54
——, spontaneous, Bilateral, 33
Poliomyelitis, Antagonism between tuberculosis and, 84
Polycythaemia vera, Tuberculosis of spleen with, 35
Polyserositis and pericarditis, Tuberculous, 33
Pomplun, F. Oxygen and carbon dioxide of arterial and venous blood in pulmonary tuberculosis, 3
Pongor, F., and Trojan, Margarethe. Costa reaction, 25
Poras, J. Circulation in tuberculosis, 2
Postoperative massive collapse and pneumonia, 70
—— —— ——, Treatment of, 71
Pottenger, F. M. Insidious and acute tuberculosis, 18
Pregnancy and tuberculosis, 85
Pressure, Pleural, after phrenicectomy, 59
Primary carcinoma of lungs, 89
—— tuberculosis of larynx, 115
—— —— —— prostate, 42
—— —— —— skin, 43
—— tuberculous infection of tonsil, 115
Prognosis of cavities, 92
—— —— tuberculosis of joints, 37
Prognostic tests, 92
Prostate, tuberculosis of, Primary, 42
Proteolytic ferments, Serum, in pulmonary tuberculosis, 1
Pseudocavity syndrome, 113
Psittacosis, 78
Pubis, symphysis, Tuberculosis of, 39
Pulmonary abscess, Treatment of, with artificial pneumothorax, 75
—— abscesses, deepseated, Treatment of, 75
—— congestion and oedema, Rapid breathing from, 78
—— diseases, Exercises in, 120
—— lesions, Asbestos fibres in, 69
—— lymphogranulomatosis, 79
—— tuberculosis, Acute respiratory infections and, 31
—— —— and sport, 46

Pulmonary tuberculosis, arterial and venous blood in, Oxygen and carbon dioxide of, 3
—— ——, Atrophy of testicles in, 42
—— ——, Calcium exchange in, 111
—— ——, Cholesterol and resistance in, 1
—— ——, complications of, Important, 28
—— ——, diagnosis of, Early, 20
—— ——, Diagnostic aids in, 20
—— ——, —— errors in, 21
—— ——, diet in, Salt-free, 47
—— ——, early, Sign of, 19
—— ——, ferments in, Serum proteolytic, 1
—— ——, Hilar reactions and, 104
—— ——, Incipiency of, 19
—— ——, latent, Influenza and, 67
—— ——, Lipatren in, 66
—— ——, Mixed infection in, 11
—— ——, Soft palate in, 29
—— ——, Treatment of, 44
—— ——, —— ——, by dust inhalation, 47
—— ——, —— ——, with helpin, 66
—— ——, —— ——, —— lipatren, 66
—— ——, —— ——, —— lopion, 65
—— ——, —— ——, —— metallosan, 64
—— ——, —— ——, —— quartz light, 61
—— ——, —— ——, —— solganol, 64
—— ——, —— ——, —— tebeform, 65
—— ——, X-ray "quality" diagnosis in, 26
—— ——, X-rays in, 26
Pulmonary tumor and tuberculosis, differential diagnosis of, Artificial pneumothorax in, 28
Puncture of chest, 60
Pus from BCG abscesses, Bacteriology of, 102
——, Reaction of, in abscesses, 2
Pyonephrosis, Tuberculous, diagnosed spondylitis, 42

"Quality" diagnosis, X-ray, in pulmonary tuberculosis, 26
Quartz light, Treatment of pulmonary tuberculosis with, 61

Rabbits, reinfected, tubercle bacilli in, Fate of, 106
Rabino, A. Haemorrhagic diathesis in tuberculosis, 103
——, ——. Pleurisy complicating artificial pneumothorax, 127
Rabinowitsch-Kempner, Lydia. Filterability of tuberculosis virus, 6
Radiation, ultraviolet, Sources of, 62
——, ——, Value and abuses of, 61
Ragolsky, H. Association of surgical and intrathoracic tuberculosis, 36
Rapid breathing from pulmonary congestion and oedema, 78
—— diagnosis of tuberculosis by animal inoculation, 114
Rawik-Scerbo, W. Bilateral artificial pneumothorax, 58
Reaction, Besredka's, in practical work, 25
——, Cholesterol agglutination, in tuberculosis, 110
——, Costa, 25
—— of lymph nodes to tuberculin, 16
—— —— pus in abscesses, 2
——, red-cell sedimentation, Lipoid treatment and, 66
——, sedimentation, Red-cell, 115
——, tuberculin, Testicular, 17
Reactions, Cellular, in tuberculosis. 103
——, Cultural and staining, of paralysis-producing bacillus, 105
——, Hilar, and pulmonary tuberculosis, 104
——, intracutaneous tuberculin, Size of, 95
——, skin, Tuberculin, 94
——, ——, Vasomotor irritability and, 2
——, Tuberculin, among hospitalized children in Iowa, 95
Rechtman, A. M. Tuberculous osteitis of knee and rib, 39
Recovery from tuberculosis, Factors in, 45
Red-cell sedimentation reaction, 115
—— —— ——, Lipoid treatment and, 66
Reed, G. B., and Rice, C. E. Pigmentation of acid-fast bacteria, 11
Reeves, R. I., and Golden, R. Calcified abdominal lymph nodes, 35
Reinfected rabbits, tubercle bacilli in, Fate of, 106
Relation of tuberculous meningitis to other forms of tuberculosis, 40
Renal tuberculosis, 41

Resistance in pulmonary tuberculosis, Cholesterol and, 1
Respiratory infections, Acute, and pulmonary tuberculosis, 31
Results of oleothorax, 57
—— —— tuberculin therapy, 66
Reumaux, L., and Bac, E. Adhesive pleuritis in artificial pneumothorax, 126
Rheumatism, Tuberculous, 36
Rib, Tuberculous osteitis of knee and, 39
Rice, C. E., and Reed, G. B. Pigmentation of acid-fast bacteria, 11
Rich, A. R. Allergy in tuberculosis, 14
Rickmann, L., and Ernst, F. Prognostic tests, 92
Riesenberg, M., and Schaffle, K. Meningitis, 40
Risks of thoracoplasty, Immediate, 125
Rist, Burman and Sergent. Sudden death following phrenicectomy in diabetic, 125
Rist, E. After-care of tuberculosis patients, 120
Robins, S. A. Fibrosarcoma of mediastinum, 91
Roentgenograms of chest in children, Interpretation of, 95
Roentgenology of bones, 28
Rôle of lung in intermediate nitrogen metabolism, 3
Rolland, J., and Maurer, A. Technique of thoracoplasty, 121, 124
Rolleston, J. D. Tracheotomy and tuberculosis, 97
Rollier, A. Pigmentation, 63
Roloff, W. Culture method of Hahn, 23
Ross, Joan, Burrell, L. S. T., and Hare, Dorothy. Tuberculous polyserositis and pericarditis, 33
Rossel, G. Pleural complications of artificial pneumothorax, 52
Rothstein, J. L., and Leopold, J. S. Miliary tuberculosis with cerebral arterial thrombosis, 119

Sachs, Erna. Tuberculosis of spleen with polycythaemia vera, 35
Sachs-Müke, and Hilgers, W. E. Complement fixation in tuberculosis, 24
Saenz, A., Calmette, A., and Valtis, J. Filterable elements of tuberculosis virus, 9
Saidman, M. Treatment of deepseated pulmonary abscesses, 75
Saleur, H., and Parisot, J. Infant mortality in tuberculous environment, 102
——, ——., Parisot, J., and Fernier, L. Bacteriology of pus from BCG abscesses, 102
Salt, Metallic, therapy, 63, 64
Salt-free diet in pulmonary tuberculosis, 47
Salt-water baths, Action of, 47
Sanatoria, Uses and limitations of, 45
Sanocrysin, 64
—— treatment, 64
Saphir, O. Liver and pancreas in tuberculosis, 34
Saponified tubercle bacilli, Immunization with, 14
Schachwuwarly, M., and Stein, W. Complement fixation in tuberculosis, 24
Schaefer, R. Mineral content of tuberculous lungs, 2
Schaffle, K., and Riesenberg, M. Meningitis, 40
Schenck, H. P., and Kern, R. A. Diagnosis of sinus disease, 67
Scheurlen, F. Serum albumen and globulin in tuberculosis, 2
Schiller, I. Cultivation of tubercle bacilli from human sputum, 23
Schlapper, K., and von Oppenkowski, H. Lipatren in pulmonary tuberculosis, 66
Schlesinger, H. Pleurisy in aged, 33
Schmitt. Physiotherapy of genitourinary tuberculosis, 117
Scholz, H. Collapse therapy in tuberculosis, 49
Schott, F. Cement dust lung, 68
Schrader, G. Tubercle bacilli in latent tuberculosis, 11
Schrag, E. Pregnancy and tuberculosis, 85
Schroeder, M. J. Miliary tuberculosis, 44
Schuberth, A. Vasomotor irritability and skin reactions, 2
Schulte-Tigges, H. Anthracosis and tuberculosis, 68
Schulz, E. Combined tuberculin and gold treatment of tuberculosis, 65
Schwalm, E. Artificial pneumothorax in differential diagnosis of pulmonary tumor and cavity, 28

Schwalm, E. Salt-free diet in pulmonary tuberculosis, 47
Schwarzmann, E., and Maendl, H. Phrenicoexairesis, 59.
Scott, W. J. M. Treatment of postoperative massive collapse, 71
Sections, histological, tubercle bacilli in, Demonstration of, 22
Sedimentation reaction, Red-cell, 115
—— ——, ——, Lipoid treatment and, 66
Sensitiveness of guinea pigs to tuberculin, 109
—— to glycerin-bouillon, 17
Sensitization, Cottonseed, 84
——, Tuberculin type of, with egg-white, 107
Sergent, E., and Stoichitza, N. Pseudocavity syndrome, 113
Sergent, Rist and Burman. Sudden death following phrenicectomy in diabetic, 125
Serous membranes, Effect of iodized oils on, 65
Serum albumin and globulin in tuberculosis, 2
—— flocculation test, 25
—— proteolytic ferments in pulmonary tuberculosis, 1
—— treatment of pneumonia, 73
Sever, J. W. Tuberculosis of spine, 38
Sevier, J. A. Treatment of pulmonary tuberculosis, 44
Sgalitzer, M. Benign tuberculous spondylitis, 38
Sherwood, M. B. Cellular reactions in tuberculosis, 103
Shock, Anaphylactic, 84
Shope, R. E., and Lewis, P. A. Cultural and staining reactions of paralysis-producing bacillus, 105
——, ——. ——., —— ——, ——. ——. Paralytic disease in guinea pigs due to tubercle bacillus, 104
Sign in artificial pneumothorax, New, 126
—— of early pulmonary tuberculosis, 19
Signs in early diagnosis, Symptoms and, 20
Sihle, M. Sign of early pulmonary tuberculosis, 19
Silicosis, 68
——, Aetiology of, 68
——, Tuberculosis and, 68
Simmons, J. S., Gardner, L. L., and St. John, J. H. Nematode infestation of lung, 79
Simulating tuberculosis, Conditions, 22
Singer, E., and Loewenstein, E. Invisible forms of tubercle bacillus, 6
Sinus disease, Diagnosis of, 67
Sisson, W. R., Vogt, E. C., and Day, H. F. Anaphylactic shock, 84
Size of consolidated lung in lobar pneumonia, 72
—— —— intracutaneous tuberculin reactions, 95
Skin as organ of immunity, 16
—— reactions, Tuberculin, 94
—— ——, Vasomotor irritability and, 2
—— tuberculosis and immunity, 44
——, —— of, Primary, 43
Smith, C. H. Tuberculin skin reactions, 94
Smith, J. K., Halliday, C. H., and Cummins, W. T. Coccidioidal granuloma and tuberculosis, 80
Soft palate in pulmonary tuberculosis, 29
Solarization of vitaglass, 62
Solganol, Treatment of pulmonary tuberculosis with, 64
Sonnenfeld, A. Mechanism of phrenicoexairesis, 59
Sources of tuberculous infection, Nursemaids and teachers as, 93
—— —— ultraviolet radiation, 62
Spine, Tuberculosis of, 38
Spleen, Tuberculosis of, with polycythaemia vera, 35
Spondylitis, tuberculous, Albee operation in, 39
——, ——, Benign, 39
——, —— pyonephrosis diagnosed, 42
Spontaneous after artificial pneumothorax, 58
—— nontuberculous pneumothorax, 77
—— pneumothorax, Bilateral, 33
Sport, Pulmonary tuberculosis and, 46
Sprungmann, E. Progress of cavities, 92
Sputum flora in aspiration pneumonia, 11
——, human, Cultivation of tubercle bacilli from, 23
St. John, J. H., Simmons, J. S., and Gardner, L. L. Nematode infestation of lung, 79

Stahnke, E. N. Treatment of surgical tuberculosis, 37
Staining reactions, Cultural and, of paralysis-producing bacillus, 105
Stein, M., and Schachwuwarly, M. Complement fixation in tuberculosis, 24
Steube, M., and Hantschmann, L. Effect of complications on colloid lability, 1
Stobie, W. Symptoms and signs in early diagnosis, 20
Stock, W. X-ray treatment of iridocyclitis, 29
Stoichitza, N., and Sergent, E. Pseudocavity syndrome, 113
Strauss, A. Treatment of tuberculosis with copper, 65
Streptococci, nonhaemolytic, Allergy to, 83
Strumia, M., Mudd, S., Mudd, E. B. H., Lucke, B., and McCutcheon, M. Antibodies in experimental tuberculosis, 13
Stuart, G. O., and Hinton, W. A. Cholesterol agglutination reaction in tuberculosis, 110
Sturm, F. Fresh tuberculous foci and carcinomatosis, 91
Subdiaphragmatic gas, 81
Sudden death following phrenicectomy in diabetic, 125
Sukiennikowa, N. Tuberculosis and malaria, 84
Surgery of lung, 51
—— —— oesophagus and lungs, 52
——, Thoracic, 50, 51
Surgical and intrathoracic tuberculosis, Association of, 36
—— tuberculosis, 117
—— ——, diagnosis of, Animal inoculation in, 37
—— ——, Treatment of, 37
—— ——, —— ——, by Finikoff's method, 117
Sutherland, C. G. Roentgenology of bones, 28
Sutton, A. C., Howard, W. T., Jr., and Pearl, R. Treatment of cancer with tuberculin, 87
Swift, H. M., and Derick, C. L. Allergy to nonhaemolytic streptococci, 83
Sylla, A. Atrophy of testicles in pulmonary tuberculosis, 42
Symphysis pubis, Tuberculosis of, 39
Symptoms and signs in early diagnosis, 20
—— of tuberculosis, Appearance of, 17
Syndrome, Pseudocavity, 113
Syphilis of lung, 81
——, Tuberculosis and, 81
Szasz, E. Allergy in tuberculosis, 15
v. Szaboky, J. Prognostic tests, 92

Taylor, M. H. Tuberculin reactions among hospitalized children in Iowa, 95
Teachers as sources of tuberculous infection, Nursemaids and, 93
Tebeform, Treatment of pulmonary tuberculosis with, 65
Technique of bronchography, 27
—— ——, thoracoplasty, 121, 122, 124
Temperature of gas in artificial pneumothorax, 54
Test, flocculation, Serum, 25
——, Perspiration, for tuberculosis, 28
——, Vernes, in tuberculosis, 25, 26
Testicles, Atrophy of, in pulmonary tuberculosis, 42
Testicular tuberculin reaction, 17
Tests, Prognostic, 92
Teutschlaender, O. Antagonism between tumors and tuberculosis, 91
Therapy, Collapse, in tuberculosis, 49
——, Metallic salt, 63, 64
——, tuberculin, Results of, 66
Thompson, St. C. Tuberculosis of larynx, 31
Thomson, A. P. Psittacosis, 78
Thoracic surgery, 50, 51
Thoracoplasty, 52
——, Anaesthesia for, 125
——, risks of, Immediate, 125
——, Technique of, 121, 122, 124
Thoracoscope, New, 125
Thoracoscopy and cauterization, 60
Thorpe, E. S., Jr. Bronchiectasis in childhood, 74
Thrombosis, arterial, cerebral, Miliary tuberculosis with, 119
Thyroid, Tuberculosis of, 30
Tonsil, tuberculous infection of, Primary, 115
Torin, J., and Bouessée, F. Oleothorax, 54

Townsend, W. G. Primary tuberculosis of prostate, 42
Tracheobronchial lymph nodes, tuberculosis of, Diagnosis of, 94
Tracheotomy and tuberculosis, 97
Transfusion for haemoptysis, Pneumothorax or, 54
Treatment, Lipoid, and red-cell sedimentation reaction, 66
—— of cancer with tuberculin, 87
—— —— deep seated pulmonary abscesses, 75
—— —— empyema, 76
—— —— exudates of artificial pneumothorax, 54
—— —— iridocyclitis, X-ray, 29
—— —— orthopaedic tuberculosis by vigantol, 37
—— —— postoperative massive collapse, 71
—— —— pulmonary abscess with artificial pneumothorax, 75
—— —— —— tuberculosis, 44
—— —— —— —— by dust inhalation, 47
—— —— —— —— with helpin, 66
—— —— —— —— —— lipatren, 66
—— —— —— —— —— lopion, 65
—— —— —— —— —— metallosan, 64
—— —— —— —— —— quartz light, 61
—— —— —— —— —— solganol, 64
—— —— —— —— —— tebeform, 65
—— —— surgical tuberculosis, 37
—— —— —— —— —— by Finikoff's method, 117
—— —— tuberculosis by limitation of fluids, 47
—— —— ——, Combined tuberculin and gold, 65
—— —— ——, Dispensary, 46
—— —— ——, Newer, 119
—— —— —— with copper, 65
—— —— —— —— ergosterol, 62
——, Sanocrysin, 64
——, Serum, of pneumonia, 73
——, X-ray, of iridocyclitis, 29
Trenti. Artificial pneumothorax in diagnosis of cavity, 28
Treves, N. Tumor of breast with tuberculous axillary adenitis, 31
Troisier, J. Hereditary tuberculous haemoptyses, 17
Trojan, Margarethe, and Pongor, F. Costa reaction, 25
Tscharny, A., and Krassowitzkaja, S. Rôle of lung in intermediate nitrogen metabolism, 3
Tube, Culture, for tubercle bacillus, 3
Tubercle bacilli, Counting of, 4
—— ——, Cultivation of, 23
—— ——, —— ——, from human sputum, 23
—— ——, Demonstration of, in histological sections, 22
—— ——, Diagnostic culture of, 22
—— ——, Effect of dyes on, 10
—— ——, Fate of, in reinfected rabbits, 106
—— —— in faeces, 107
—— —— —— latent tuberculosis, 11
—— ——, Microscopic determination of, 22
—— ——, saponified, Immunization with, 14
—— ——, virulence of, Determination of, 10
—— bacillus, Antigenic substances of, 12, 13
—— ——, Biology of, 5
—— ——, carbohydrate antigen of, Anaphylaxis with, 108
—— ——, Culture tube for, 3
—— ——, Cytology and microchemistry of, 4
—— ——, Filterability of, 9
—— ——, Filterable forms of, 7
—— ——, Invisible forms of, 6
—— ——, Paralytic disease in guinea pigs due to, 104
—— ——, Variability of, 5
Tuberculin allergy and antituberculosis immunity, 109
—— and gold treatment of tuberculosis, Combined, 65
——, Effect of fermentation on, 16
——, Haemoclastic crisis and, 114
——, Reaction of lymph nodes to, 16
—— ——, Testicular, 17
—— reactions among hospitalized children in Iowa, 95
—— ——, intracutaneous, Size of, 95
——, Sensitiveness of guinea pigs to, 109
—— skin reactions, 94
—— therapy, Results of, 66
——, Treatment of cancer with, 87

Tuberculin type of sensitization with egg-white, 107
Tuberculosis, Action of nonspecific irritants or blood in, 66
——, activity of, Diagnosis of, 21
——, Allergy in, 14
—— and malaria, 84
—— —— nervous system, 40
—— —— poliomyelitis, Antagonism between, 84
—— —— silicosis, 68
—— —— syphilis, 81
——, Antagonism between tumors and, 91
——, Anthracosis and, 68
——, Cellular reactions in, 103
——, Cholesterol agglutination reaction in, 110
——, Circulation in, 2
——, Coccidioidal granuloma and, 80
——, Codliver oil in, 63
——, Coincident malignant tumor and, 87
——, Collapse therapy in, 49
——, Complement fixation in, 24
——, Conditions simulating, 22
——, diagnosis of, Rapid, by animal inoculation, 114
——, Diet in, 46
——, experimental, Antibodies in, 13
——, ——, Diminished air-pressure and, 3
——, Failure to diagnose, 22
——, feeding in, Basic, 46
——, genitourinary, Physiotherapy of, 117
——, Haemorrhagic diathesis in, 103
——, Hilum, 32
——, Immunity to, 16
—— in infants, Milk and, 93
—— —— interior East Africa, 92
——, Insidious and acute, 18
——, Intestinal, 34
——, Laryngeal, 30
——, latent, Tubercle bacilli in, 11
——, Liver and pancreas in, 34
——, lymph-node, Mesenteric, 35
——, metabolism in, Carbohydrate, 1
——, Miliary, 44
——, ——, with cerebral arterial thrombosis, 119
—— mortality in Belgium, 92
——, Observations on, 22
—— of aorta, 33
Tuberculosis of infants and children, Monocytes in, 96
—— —— joints, Prognosis of, 37
—— —— larynx, 31
—— —— ——, Primary, 115
—— —— mesenteric lymph nodes, Codliver oil and butter in, 35
—— —— prostate, Primary, 42
—— —— skin, Primary, 43
—— —— spine, 38
—— —— spleen with polycythaemia vera, 35
—— —— symphysis pubis, 39
—— —— thyroid, 30
—— —— tracheobronchial lymph nodes, Diagnosis of, 94
—— —— uterine adnexa, 43
——, orthopaedic, Treatment of, by vigantol, 37
—— patients, After-care of, 47, 120
——, Perspiration test for, 28
——, Pregnancy and, 85
——, proteolytic, ferments in, Serum proteolytic, 1
——, pulmonary, Acute respiratory infections and, 31
——, ——, and sport, 46
——, ——, arterial and venous blood in, Oxygen and carbon dioxide of, 3
——, ——, Atrophy of testicles in, 42
——, ——, Calcium exchange in, 111
——, ——, Cholesterol and resistance in, 1
——, ——, complications of, Important, 28
——, ——, diagnosis of, Early, 20
——, ——, Diagnostic aids in, 20
——, ——, —— errors in, 21
——, ——, diet in, Salt-free, 47
——, ——, early, Sign of, 19
——, ——, Hilar reactions and, 104
——, ——, Incipiency of, 19
——, ——, latent, Influenza and, 67
——, ——, Lipatren in, 66
——, ——, Mixed infection in, 11
——, ——, Soft palate in, 29
——, ——, Treatment of, 44
——, ——, —— ——, by dust inhalation, 47
——, ——, —— ——, with helpin, 66
——, ——, —— ——, —— lipatren, 66
——, ——, —— ——, —— lopion, 65

Tuberculosis, pulmonary, Treatment of, with metallosan, 64
—, —, — —, — quartz light, 61
—, —, — —, — solganol, 64
—, —, — —, — tebeform, 65
—, pulmonary tumor and, differential diagnosis of, Artificial pneumothorax in, 28
—, pulmonary, X-ray "quality" diagnosis in, 26
—, —, X-rays in, 26
—, recovery from, Factors in, 45
—, Relation of tuberculous meningitis to other forms of, 40
—, Renal, 41
—, Serum albumen and globulin in, 2
—, Skin, and immunity, 44
—, Surgical, 117
—, — and intrathoracic, Association of, 36
—, —, diagnosis of, Animal inoculation in, 37
—, —, Treatment of, 37
—, —, — —, by Finikoff's method, 117
—, symptoms of, Appearance of, 17
—, Tracheotomy and, 97
—, Treatment of, by limitation of fluids, 47
—, — —, Combined tuberculin and gold, 65
—, — —, Dispensary, 46
—, — —, Newer, 119
—, — —, with copper, 65
—, — —, —, ergosterol, 62
— virus, Filterability of, 6
— —, Filterable elements of, 9
Tuberculous axillary adenitis, Tumor of breast with, 31
—, Comparison of, with other infections, 29
— environment, Infant mortality in, 102
— epididymitis, Curettage in, 42
— foci, Fresh, and carcinomatosis, 91
— haemoptyses, Hereditary, 17
— infection of tonsil, Primary, 115
— —, sources of, Nursemaids and teachers as, 93
— laryngitis, Benzyl cinnamic ester in, 30
— lungs, Mineral content of, 2
Tuberculous meningitis, Diagnostic difficulties in, 119
— —, Relation of, to other forms of tuberculosis, 40
— — with unusual cerebrospinal fluid, 40
— osteitis of knee and rib, 39
— polyserositis and pericarditis, 33
— pyonephrosis diagnosed spondylitis, 42
— rheumatism, 36
— spondylitis, Albee operation in, 39
— —, Benign, 38
Tumor, malignant, and tuberculosis, Coincident, 87
— of breast with tuberculous axillary adenitis, 31
—, pulmonary, and tuberculosis, differential diagnosis of, Artificial pneumothorax in, 28
Tumors and tuberculosis, Antagonism between, 91
—, Intrathoracic, 85
—, —, diagnosis of, X-ray, 87
— of lung, Malignant, 86
Turnbull, H. M., and Konstam, G. L. S. Ayerza's disease, 81
Turpin, R., and Weill-Hallé, B. Immunization with BCG, 99

Ultraviolet radiation, Sources of, 62
— —, Value and abuses of, 61
Unusual cerebrospinal fluid, Tuberculous meningitis with, 40
Use of BCG in Belgium, 101
Uses and limitations of sanatoria, 45
Uterine adnexa, Tuberculosis of, 43
Uyei, N., and Corper, H. J. Diagnostic culture of tubercle bacilli, 22

Valtis, J., Saenz, A., and Calmette, A. Filterable elements of tuberculosis virus, 9
Value and abuses of ultraviolet radiation, 61
Van Beneden, J. Use of BCG in Belgium, 101
Variability of tubercle bacillus, 5
Vasomotor irritability and skin reactions, 2
Veiel, F. Climate and lupus vulgaris, 44
Venous blood, arterial and, in pulmonary tuberculosis, Oxygen and carbon dioxide of, 3

Vernes test in tuberculosis, 25, 26
Viets, H. R., and Watts, J. W. Tuberculous meningitis with unusual spinal fluid, 40
Vigantol, Treatment of Orthopaedic tuberculosis by, 37
Virulence of tubercle bacilli, Determination of, 10
Virus, tuberculosis, Filterability of, 6
——, ——, Filterable elements of, 9
Vitaglass, Solarization of, 62
Vogt, E. C., Day, H. F., and Sisson, W. R. Anaphylactic shock, 84
von Daranyi, J. Serum flocculation test, 25
von Oppenkowski, H., and Schlapper, K. Lipatren in pulmonary tuberculosis, 66

Wachter, R., and Groedel, F. M. X-ray "quality" diagnosis in pulmonary tuberculosis, 26
Walbum, L. E. Metallic salt therapy, 64
Walsh, E. L., and Crandall, L. A. Effect of iodized oils on serous membranes, 65
Warlimont, Erna. Sanocrysin, 64
Wasson, W. W. Bronchosinusitis disease, 67
Watts, J. W., and Viets, H. R. Tuberculous meningitis with unusual spinal fluid, 40
Webb, G. B. Early diagnosis of pulmonary tuberculosis, 20
Weichardt, W. Immunity to tuberculosis, 16
Weidman, F. D. Pathogenic fungi, 82
Weill-Hallé, B., and Turpin, R. Immunization with BCG, 99
Weinbren, M. Value and abuses of ultraviolet radiation, 61
Werner, A. J., and Grumbach, A. Besredka's reaction in practical work, 25
White, J. S., and Boyes, C. R. Codliver oil in tuberculosis, 63
Wiening, H., and Blumenberg, W. Counting of tubercle bacilli, 4
Wildre, D., and Borock, M. Air embolism in artificial pneumothorax, 58
Williams, C., and Budd, S. W. Tuberculosis of thyroid, 30
Williams, L. R., and Hill, A. M. Appearance of symptoms of tuberculosis, 17
——, ——. ——., —— ——, —— ——. Diagnostic aids in pulmonary tuberculosis, 20
Wissing and Henius, K. Treatment of pulmonary tuberculosis with lopion, 65
Withington, P. R. Milk and tuberculosis in infants, 93
Wood, W. B. Asbestosis, 69
Wounds, chest, Penetrating, 67

X-ray diagnosis of intrathoracic tumors,
—— features of massive collapse, 71
—— findings of heart, 82
—— image of aspiration pneumonia, 28
—— "quality" diagnosis in pulmonary tuberculosis, 26
—— treatment of iridocyclitis, 29
X-rays in pulmonary tuberculosis, 26

Young, R. A. Thoracic surgery, 50

Zaaijer, J. H. Surgery of oesophagus and lungs, 52
Ziegler, O. Immunity to tuberculosis, 16
Ziehl-Neelsen method preferred, 22